Johne's Disease: Environment And Management*

PART II — CONTROL AND PREVENTION

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This presentation on Johne's disease, the second of a two-part discussion, is a searching analysis of factors relating to this important malady of dairy cattle. The first part (General Considerations) appeared in VETERINARY MEDICINE for November 1952.

WITH the knowledge of Johne's disease, as discussed in part I, veterinary practitioners and livestock owners can make substantial and effective progress in preventing, controlling, and ultimately eliminating it from a herd of cattle.

Johne's disease requires a specific, detailed stable hygiene and herd management that is far more exacting than that needed for tuberculosis prevention. It should also be remembered that once an effective program is put into operation it wi'l not only reduce materially the incidence of Johne's disease, but will markedly decrease the incidence of many other infectious diseases common in cattle housed or maintained in rather close confinement.

In protecting the noninfected herd, it is only necessary to remember how the infection may be introduced. Conceivably, the bacillus could be carried from an infected manure pile to a noninfected farm by birds, other animals, and farm drainage; but the two main avenues of introduction for Johne's disease infection are: (1) newly purchased animals harboring the infection; (2) cattle trucks that have transported infected animals.

In undertaking to put the disease under control in a herd already infected, it is necessary to consider the following: (1) isolation of suspected animals; (2) the material in and the type of stable construction; (3) manger construction; (4) feeding practices; (5) disposition of manure; (6) the use of stable utensils; (7) footwear of attendants; (8) water supply and drainage; (9) location of calf pastures; (10) bleeding cows for blood tests.

Of all these, the isolation of suspected animals, the type of manger, and the feeding practices are the most important.

The Noninfected Herd

Origin of the infection in a noninfected herd is almost always the result of a purchased animal harboring the infection. The Johne's bacillus is bought and sold.

In areas where commercial truckers haul cattle from the farm to the sales yards, and from the sales yard to the farm, they sometimes haul infected cattle. The truck itself may become contaminated. Healthy cattle hauled in such trucks might easily carry the infection onto a farm. Infected manure and straw in the truck may become scattered on the ground when loading and unloading.

1. Newly Arrived Animals. It cannot be over-emphasized that Johne's disease is most often, if not always, introduced into a clean herd by an infected animal. Therefore, it is self-evident that prevention must be based on precautions one can take in purchasing additions to the herd, and, even more to the point, in handling newly purchased animals.

Documented records reveal that the infection may be present in herds that are, for all

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practical purposes, home-bred. The time the disease was introduced may be unknown in many cases, although in some infected herds, largely home-bred, the time and origin are known or can be determined from the herd records.

To assume that any purchased, apparently healthy animals, are disease-free is unwise. In the opinion of the owner or purchaser, a herd may be entitled to be considered a Johne's disease-free herd. However, the long period of incubation, the chronic nature of the disease, often the long interval between cases, and the absence of a positive diagnosis of former cases of like sicknesses, all contribute to the likelihood that the disease may be present and undetected. In some instances, a detailed study of the herd record of animal losses in the preceding two years might reveal that one or more animals had shown symptoms of chronic diarrhea and loss of flesh, and had either been disposed of or died. In such cases, the possibility of Johne's disease should be given full consideration.

It is therefore advisable to hold in quarantine, for some days or weeks, any animal newly arrived to the farm.

Cows in advanced pregnancies should be observed closely, preferably held in quarantine, for some weeks after they have freshened, since infected cows will frequently develop symptoms of diarrhea and loss of flesh within a few days or weeks after parturition.

Any animal on the farm, part of the herd or newly arrived to the herd, should immediately be put in quarantine if it develops diarrhea.

The period between primary infection and development of symptoms is often so long as to make quarantine a difficult and unsatisfactory procedure. However, it should be possible to hold in isolation the cow that is to freshen shortly after purchase, and the cow that has freshened shortly before purchase. If a herd has been recently assembled, or new arrivals have been added to it, the possibilities of the presence of Johne's infection are considerably increased, and quarantine becomes doubly important.

Where quarantine is neither possible nor practical, there are two other procedures which can be followed. There should be, first of all, a microscopic examination of the feces, following the technics that have been developed for recognition of the Johne's bacillus in the feces and in the mucus that may be obtained from the rectal membrane. At the same time, the feces should be examined for Coccidia and for the presence of stomach worm eggs and other forms of parasitism in cattle.

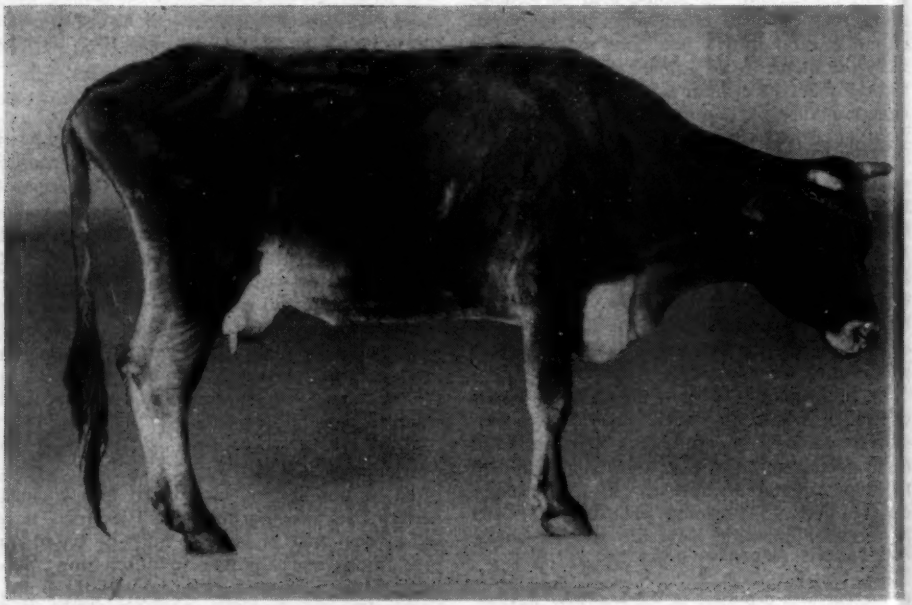

Fig 1. This cow was out of a herd where 15 to 20 cows developed and died of Johne's disease during a five-year period. The first case on the farm, reported to have had all the characteristic symptoms of Johne's disease, was an imported cow that died in 1916.

If the feces or the mucus from the membrane shows the presence of Johne's bacilli, the animal should immediately be quarantined and disposition made of it. It may be killed and buried, or killed and used for tankage. If not too thin in flesh, the animal can be sold for slaughter. Under no condition should it be sold, either privately or on the open market, for transfer to another farm. Unfortunately, some cows with Johne's disease are sold and purchased on the open market, occasionally the same animal two or three times.

If there is no evidence of the bacilli from these examinations, bearing in mind the long incubation period, the cow should be watched carefully for any change in the consistency of the manure and frequency of defecation. It is customary, when a cow develops diarrhea, to have her treated. It should be remembered, however, that following the first, second, or third attack of diarrhea due to Johne's disease, the diarrhea will subside in the course of a few days, regardless of whether the animal was or was not treated.

Early attacks of diarrhea are intermittent. In the first attack the feces are soft and mushy and are usually again quite normal after a day or two. The second attack will often occur within a few days, the feces are more evidently soft, and remain so for a longer period. Following the second, and especially the third attack, the consistency of the excrements becomes fluid, is persistent, and the tail and body of the cow become soiled.

Any animal that has a second attack of diarrhea within a matter of days following the first attack should certainly be put in quarantine until a diagnosis has been made.

2. *Cattle Trucks.* Effective sanitary control of trucks for commercial trucking of cattle is a problem in itself. There are, in the United States, many places where public auction sales are held weekly, and many of the animals coming to and going from these sales are hauled by commercial truckers. Another agent in the spread of Johne's disease is the man who buys, sells, and trucks animals. This kind of traffic means that the same animal goes from yard to yard, and not infrequently from farm to farm. Johne's and many other diseases are spread in this manner from one area to another. However, no really effective solution to this aspect of the disease problem has ever been devised, and at present this part of the disease-prevention program seems to rest on the integrity of the primary owner of the animal as to its condition when offered for sale and disposition. All Johne's disease-infected cattle should be sold only for immediate slaughter.

Considering these factors, it is of prime importance that the owner consider the possibility of every new animal being a carrier of Johne's disease. When and if an accurate diagnostic agent is available, one that will detect earliest cases, the further precautionary measures given below may not be essential. Until that time, however, these are highly important to avoid the damage and loss that occurs when one has failed to prevent the introduction of the infection into the disease-free herd. In an infected herd, where the problem is to control and eliminate the disease already present, these further measures *must* be undertaken.

The Infected Herd

The essential problem of control and elimination of Johne's disease in a herd is that of controlling and eliminating every possible means by which stable manure could find its way into the manger, feed, water, and grazing areas—particularly into small lots and paddocks.

Since manure from an infected animal is the origin of the Johne's bacilli, every little spatter of manure is a possible source of infection. If other animals lick or nose the soiled tail and rear quarters of an infected cow, they stand a good chance of taking the infection into the mouth. It is difficult to insure that not one manure-soiled object is accidentally or negligently transferred from the rear of the cows to the front feeding area, but the number of such incidents can at least be reduced to a minimum. Never forget that the bacilli are eliminated from the infected animal only in the droppings, and that the animal becomes infected only through the mouth. The effort, therefore, in all these measures is to break the cycle between the excrements and those things that the animal takes orally.

1. Isolation of Suspected Animal. This topic has been discussed in the foregoing under The Noninfected Herd. However, since the Johne's bacillus is expelled from the infected animal largely, if not only, in the excrements, it should be self-evident how important it is to confine the cow so that the manure can be properly handled and not spread in the stable, yards, or small lots where cattle are kept.

2. Material in and Type of Stable Construction. Most stables are of concrete construction. This make possible effective cleansing, especially where there is sufficient water pressure.

3. Manger Construction. No dairy barn should ever be constructed with a sweep-in manger; it is one of the most potent means by

which Johne's bacilli and many other infections of the alimentary tract are acquired.

Sweep-in mangers are a virtual sewer system for spreading the Johne's bacilli and other infectious organisms, including those causing brucellosis, coccidiosis, tuberculosis, salmonellosis, and many others that are capable of causing intestinal disorders. *Vibrio fetus* may well be an alimentary infection; and certainly the nails, pieces of wires, and other metallic bodies found in the reticulum, and often resulting in traumatic pericarditis, can easily get into a sweep-in manger.

Those who manufacture dairy stable fixtures have taken it upon themselves to design dairy barn construction, and this construction has featured the sweep-in manger. Our economists have published many articles on labor and time-saving operations for handling a dairy herd, and certainly conveniences and improved facilities are to be encouraged. But they should not be put into operation if they jeopardize the health of the herd. The loss of one good cow may more than offset all the theoretical economies figured out on paper, and procedures that appear to be economical may not always, in the long run, result in added profits.

All dairy barns should have concrete mangers with a high front, and be of a width to prevent grain from the cows' mouths from falling out onto the floor. This will help avoid any tendency to throw back in the manger any feed from the space in front of the manger which may have become contaminated.

4. Feeding Practices. Of the several general practices in feeding, there are three that deserve special mention.

The value of a high, wide manger as compared with the sweep-in manger has already been emphasized.

The rest-shed, milking-parlor method of handling dairy cows in milk, where the grain is fed at time of milking, will help control manger contamination quite as effectively as the manger with the high front. Hay fed in the rest-shed should be in racks. If silage is fed in the rest-shed, the mangers should be protected as much as possible from contamination by excrements.

Attendants wearing the same shoes or boots when walking in the feeding alley, in the feed room, handling hay, and especially when forking silage out of the silo, are all possible sources of infection. In short, any object coming in contact with the excrements of a cow disseminating the Johne's bacillus, and then coming in contact with the feed or receptacle from which the animal eats, is always a very real means of spreading the infection from cow to cow in infected herds.

5. Disposition of Stable Manure. It is essential that stable manure be stored where animals cannot come into contact with it. Further, the drainage from the manure pile should not be permitted to collect in puddles, drain into streams, or drain onto grass areas where calves and cattle graze. A concrete pit with a roof is the best manure-storage bin.

6. The Use of Stable Utensils. Stable utensils used to clean behind the cows, such as shovels, forks, hoes, brooms, wheelbarrows, and tools of any kind, should not be used to clean, sweep, or handle feed either in the feed rooms or that portion of the barn in front of the mangers. Where this is permitted, the owner is helping to infect his own herd.

7. Footwear of Attendants. The shoes and overboots worn by attendants working in the rear of the cows often become soiled with the excrements. What appears to be a perfectly clean boot may easily carry thousands of bacteria, including Johne's bacilli, if any of the cows are disseminators.

Therefore, when there is, or has been, a case of Johne's disease in the herd, great care should be taken not to go with manure-soiled shoes from behind the cows to the feed room, walk on hay being fed to the cows, or go into the silo. It is not a simple matter to prevent carrying a spatter of manure on one's shoes, boots, or clothing. However, to reduce the probability of contamination, it might be well to wear rubber overshoes when milking, or cleaning the stable. These overshoes can then be removed before the attendants go to another part of the stable or barn—particularly to the areas where feed is stored and handled. Rubber overshoes could be washed before leaving the contaminated areas, or a shallow pan of antiseptic can be provided to step in. These procedures are troublesome and time-consuming; but they are necessary if the spread of the disease in a herd is to be controlled.

8. Water Supply and Drainage. It has already been emphasized that drainage from stored stable manure must be away from any area into which the animals are likely to go. Stables, grazing areas, small lots and paddocks must be arranged so that the surface water from any one of them does not drain into any of the others. This procedure will help considerably in limiting, and gradually eliminating, the contamination in any of the areas.

If there is an ample supply of water having considerable pressure, and if stable drainage is away from other used areas, it becomes possible to wash the stable daily. This, with the other measures enumerated, will reduce the

possibility of new cases of infection to a minimum, even in heavily infected herds.

9. Location of Calf Pastures. Calves should be raised in a barn separate from the cows, and the caretakers working in the cow barn should not go into the calf barn with soiled shoes. No stable utensils used in the cow barn should be used in the calf barn.

The calf pen should be of a type and construction to be easily cleaned. The feeding should be in receptacles (buckets and mangers) that can be kept clean and free of contamination by any material from the main stable.

The calf lot must be located so that it receives no drainage from the cow stable or from stored manure. In fact, the calf from birth to maturity should be kept absolutely separated from the main herd and stable.

10. Bleeding of Cows. Johne's bacilli can be spread in the process of bleeding cows. If the cow is being bled while stanchioned and the attendants holding the animal or bleeding her, stand in the manger, the Johne's bacilli can be carried into the manger on the soiled footwear. If it is necessary to stand in the manger, the footwear used when cleaning the stable or tramping through the yards and around manure piles should be removed or changed.

Conclusions

When a case of Johne's disease remains in a stable for some time before a diagnosis has been made and the animal removed, the contamination of the farm may be widespread and heavy. In such cases, it requires a specialist in farm hygiene to make a careful survey of the farm and general environment. The specialist can then determine just what detailed measures in stable construction and everyday management are necessary. The testing of all animals with johnin or an examination of the feces, particularly of the suspicious cases, would be advisable. The johnin test, however uncertain in results, would in most herds furnish information of some value for designing a program of control and prevention within the herd. To manage the herd solely on the basis of the johnin tests will not give effective results. However, when these tests are used in conjunction with the other essential procedures of diagnosis and management, they may well be of considerable help.

The procedures outlined under Control and Prevention would be, on some farms, simple and inexpensive to execute. On others, because of stable construction and the established arrangement of buildings and surrounding areas, it might be found rather difficult to work out a plan that would effectively eliminate all the major or minor possible sources of spread of infection. Whatever the situation, favorable or unfavorable, control measures are worth the time, effort, and money involved because a disease-free herd is a sound economic investment. These suggested procedures will unquestionably reduce the incidence of many of the diseases and infections of cattle although they are primarily designed for the control of Johne's disease.

Johne's disease is serious; (1) because it practically always results in the death of the animal, (2) because cattle with the disease are continuous spreaders of the bacillus in great numbers, (3) because of the great difficulty of destroying the bacillus in the excrements of infected animals, (4) because infected nonclinical cases may be sold to go into a noninfected herd, and (5) because clinical cases do find their way into the open market.

Controlling the sale of infected animals, except for immediate slaughter, would help to prevent new centers of infection. A livestock owner, reasonably familiar with the nature of the disease, and with the help and advice of his veterinarian, can effectively protect his own herd; and perhaps this is the solution to the Johne's disease problem. However, it does seem that the livestock owners, veterinarians, state livestock officials, and local livestock agencies, might, on a cooperative basis, do what they can to prevent traffic in disease-infected cattle. This would help to limit the possibilities of spread by those individuals who buy and sell cattle on a small scale.

A less common but equally serious means of spreading the disease results from the sale of apparently healthy animals that are to be used as breeding stock, but that have been bred and raised in infected herds. In badly infected herds, where losses have been sufficient in numbers to make the herd unprofitable, the owner, either on his own initiative or on the advice of his veterinarian, may dispose of the entire herd. While it may be advisable to sacrifice the herd, all the animals in it should go for immediate slaughter since many of them, apparently healthy, may be in the early stages of the incubation period of Johne's disease. These infected cattle may later break with the disease and establish a new center of infection.

All of the material in this article is based on the knowledge gained from a personal study and observation of the disease, over a period of 32 years and in many herds and

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Controlled Experiment To Test Duration Of Immunity Against Hog Cholera Induced In Pigs Vaccinated With Sterile Tissue Cultured Vaccine And Serum

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UNDER field conditions, it is conceivable that vaccinal immunity could occasionally be enhanced by exposure to hog cholera while the animals are carrying sufficient vaccinal immunity to withstand that exposure. In order to establish the duration of immunity produced in pigs vaccinated with viracine® and hog cholera antiserum injected simultaneously, an experiment was designed to obtain information under controlled conditions to supplement that obtained by mass use of the method under field conditions[1]. Under field conditions, data on immunity duration beyond the usual market age of hogs are rather difficult to obtain. This experiment was designed to determine duration of vaccinal immunity over a period of at least one year.

The dose of viracine used in this experiment was 2 cc. of the restored dried vaccine. The dose of serum administered simultaneously was varied to point out any differences in vaccinal immunity produced with 10 cc. of serum administered with the vaccine as compared with 15 cc.

All pigs used were from one ranch and were received at the hog cholera laboratory on October 4, 1951. They were held in isolated quarantine until used for the experiment. The average weight of the pigs was 65 lb. These animals were treated and ear-marked on October 11 as indicated in table below.

The two susceptible controls succumbed to virulent virus challenge, indicating that the pigs used for the experiment were susceptible to hog cholera infection.

No reactions were evident in any of the vaccinates, serum controls, or susceptible controls in contact with the vaccinates for 39 days following the start of the experiment. At this time, four of the contact controls were selected from the group and held at the laboratory. The remainder were moved to a ranch and held isolated from contact with any other animals. There were no swine on these premises.

*Cutter Laboratories, Berkeley, Calif.

®A special license to produce and market viracine commercially has been issued to Cutter Laboratories, Berkeley, Calif., by the Bureau of Animal Industry, USDA, Washington, D. C.

Number Pigs	Purpose	Dose Serum	Dose of viracine 1:50 dilution of dried residue. Culture 132051 (Same dose used in field experiments.)	Remarks
6	Serum controls	10 cc.	None	Held in barn used for normal suspectible pigs
6	Serum controls	15 cc.	None	" " "
17	Vaccinates	10 cc.	2 cc.	Held in isolated quarters
16	Vaccinates	15 cc.	2 cc.	Held in isolated quarters
8	Contact controls	——	——	In contact with vaccinates
2	Susceptible	None	None	
	Received virulent virus to determine susceptibility of the group			

At this time, six susceptible pigs from another source were added to the group to serve as susceptible controls for the purpose of indicating whether or not the group was exposed to hog cholera at any time when they were being held at the ranch.

The four contact controls removed from the group and held at the laboratory were challenged with virulent hog cholera virus on November 19, 1951. All four developed typical symptoms of hog cholera and at autopsy exhibited the usual pathological changes typical of that disease.

On December 14, 1951 (64 days after the experiment was started), a portion of each group of pigs treated was brought in from the ranch and challenged with 3 cc. of virulent hog cholera virus. The results are tabulated below:

Number Pigs	Treatment Received in October 1951	Results of Virus Challenge 64 days Postvaccination
2	10 cc. serum only	One pig had febrile reaction, sickened and recovered One pig succumbed with typical cholera syndrome
2	15 cc. serum only	Both pigs had febrile reaction but no visible clinical reaction
2	10 cc. serum with 2 cc. viracine 132051	No febrile nor visible reactions—immune
2	15 cc. serum with 2 cc. viracine 132051	No febrile nor visible reactions—immune
2	Susceptible controls	Succumbed to challenge with typical syndrome of H. C.

On February 13, 1952 (126 days after the experiment was started), another group of pigs was challenged by injection of virulent hog cholera virus. The results of this challenge are tabulated below:

Number Pigs	Treatment Received in October 1951	Results of Virus Challenge 126 days Postvaccination
2	10 cc. serum only	Susceptible
2	15 cc. serum only	Susceptible
5	10 cc. serum with 2 cc. viracine 132051	Immune
5	15 cc. serum with 2 cc. viracine 132051	Immune
2	Susceptible controls	Susceptible

Since the pigs that received 10 cc. and 15 cc. of antiserum alone at the start of the experiment were shown to be susceptible at this time, the remaining two pigs on each serum dose level were removed from the experimental group at the ranch and sold.

On April 16, 1952 (188 days after the experiment was started), the following pigs were removed from the ranch group and challenged by injection of virulent hog cholera virus with the following results:

Number Pigs	Treatment Received in October 1951	Results of Virus Challenge 188 days Postvaccination
5	10 cc. serum with 2 cc. viracine	Immune
4	15 cc. serum with 2 cc. viracine	Immune

At this time, no controls were injected, but were retained to stay with the remaining pigs in each of the two vaccinated groups for exposure at one year postvaccination.

After the vaccinated pigs in both groups were found to be immune at six months postvaccination, it was decided to obtain further information on immunity induced. In the remaining group, there were five sows held for virulent virus challenge at one year postvaccination. It was decided to have these sows bred to farrow in early October of 1952. At the time sows were challenged to check immunity at one year postvaccination, their pigs could also be challenged to determine whether or not they had been passively immunized as a result of nursing vaccinated sows. A boar was placed with the sows on June 23, 1952, after a suitable quarantine period.

Three sows farrowed October 9, 10, and 11. These with their litters were challenged on October 24, 1952, when the pigs were two weeks of age. Each sow was injected with 3 cc. of virulent virus and each baby pig with 1 cc. Another sow farrowed October 21, 1952. It was decided to wait until this litter was one month of age before the sow and litter were exposed.

The table shown on the following page summarizes the results of virulent virus challenge of the experimental animals at one year postvaccination, including the results of the baby pig exposures.

Summary

This experiment was designed to test duration of immunity produced in pigs following vaccination by injection of 2 cc. of viracine and a simultaneous dose of 10 cc. or 15 cc. of hog cholera antiserum. Controls were used to

permit interpretation of results when test challenges were made at appropriate intervals by excluding passive immunity from serum, and any possible boost of immunity from field exposure between vaccination and challenge. Susceptible animals were placed with the vaccinates to check on any possibility of the vaccinated animals shedding virus.

Sows in the vaccinated groups held for exposure at one year postvaccination were bred to farrow before challenged. Each sow and each pig in its litter was challenged by injection of virulent hog cholera virus at the same

2. There was no shedding of virus from vaccinates, as indicated by the susceptibility of unvaccinated animals held in the same pens with the vaccinates for 39 days after vaccination.

3. Serum controls that received 10 cc. and 15 cc. of serum alone were fully susceptible to hog cholera when challenged at 4½ months after vaccination. It was odd, however, that two pigs which received 15 cc. of serum alone and were challenged at 64 days after injection were not fully susceptible.

4. Pigs vaccinated with viracine (2 cc.) and

Pig Number	Treatment Received October 11, 1952	Date of Virulent Virus Challenge	Results of Challenge One Year Postvaccination
374	10 cc. serum—2 cc. viracine	10-16-52 3 cc.	Immune (no reaction)
372	10 cc. serum—2 cc. viracine	10-16-52 3 cc.	Immune (no reaction)
370	15 cc. serum—2 cc. viracine	10-16-52 3 cc.	Immune (no reaction)
371	15 cc. serum—2 cc. viracine	10-16-52 3 cc.	Immune (no reaction)
373	15 cc. serum—2 cc. viracine	10-16-52 3 cc.	Immune (no reaction)
*382	15 cc. serum—2 cc. viracine	11-10-52 3 cc.	Immune (no reaction)
375	Pen contact control placed with vaccinates at start of experiment	10-16-52 3 cc.	Susceptible
376	Pen contact control placed with vaccinates at start of experiment	10-16-52 3 cc.	Susceptible
369	None. Placed with group when moved to ranch for holding	10-16-52 3 cc.	Susceptible
377 (sow)	10 cc. serum—2 cc. viracine	10-24-52 3 cc.	Immune
2-wk.-old litter of 377—5 pigs	No treatment but nursing mother	10-24-52 1 cc.	All were immune—no reaction
378 (sow)	10 cc. serum—2 cc. viracine	10-24-52 3 cc.	Immune
2-wk.-old litter of 378—7 pigs	No treatment but nursing mother	10-24-52 1 cc.	Two pigs drowned in trough first night after challenge; five others showed no reaction
381 (sow)	10 cc. serum—2 cc. viracine	11-17-52 3 cc.	Immune
1-month-old litter of 381—8 pigs	No treatment but nursing mother	11-17-52 1 cc.	All were immune—no reaction
379 (sow)	15 cc. serum—2 cc. viracine	10-24-52 3 cc.	Immune
2-wk.-old litter of 379—7 pigs	No treatment but nursing mother	10-24-52 1 cc.	All were immune—no reaction

*There was some doubt as to whether or not this sow was pregnant. The animal was not exposed with the others but was held over until it was ascertained to be nonpregnant, then challenged.

time to test passive immunity acquired from mother's milk. Three sows and their litters were challenged when the pigs were two weeks of age; one sow and litter when the pigs were four weeks of age.

The result of this controlled duration of immunity experiment justifies the following conclusions:

1. No reactions occurred following viracine and serum vaccination with either dose of serum when administered simultaneously.

serum at either 10 cc. or 15 cc. dose levels were found to be solidly immune to virulent hog cholera virus injection at two months, 4½ months, six months, and one year postvaccination.

5. Suckling pigs, two weeks and four weeks of age, nursing their dams which had been vaccinated with viracine and serum one year earlier, were found to be solidly immune as

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Winter Dysentery In Dairy Cattle

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WINTER dysentery occurs as a herd-wide diarrhea among stabled dairy cattle. During each of the past three years, first cases were observed on November 14. The disease has not been observed in cattle on pasture.

Outbreaks of winter dysentery are characterized by diarrhea, a high percentage of morbidity, and a drop of 50% or more in milk production. Mortality rate is low. The chief concern of the veterinarian dealing with outbreaks is to restore normal milk production promptly.

Etiology

The cause of this disease has been attributed to organisms of the genus Vibrio[1]. Veterinarians cite evidence that the causative agent is infectious and contagious. They occasionally have been called to treat the disease before an appreciative but not germ-conscious audience of neighbors and then, shortly, found themselves treating similar outbreaks in the herds of these onlookers. The infection appears to spread easily from herd to herd, often, perhaps, on contaminated clothing and footwear. This demands that the practitioner demonstrate his knowledge and skill in the disinfection of personal equipment and apparel.

Abrupt change of feed appears to be a predisposing factor in the majority of cases. Such a change occurred in the herd of a client who tired of cooking cull navy beans for his cattle. Advised by his county agent that ground, dry beans could be fed raw as no more than 20% of a grain mixture, the farmer switched his milking cows in the course of one feeding to such a dry mixture. Cooked beans had previously been the only supplement to the herd's roughage ration. Winter dysentery attacked this herd four or five days after the change. The owner's wrath toward his county agent subsided when it was pointed out that heifers and dry cows not getting the new ration developed the disease 48 to 72 hours after its appearance in the herd.

Abrupt changes in feed could predispose to the disease in at least two different ways. First, the change might serve to mechanically carry the causative agent to the herd, as on contaminated bags or feed. Second, if the causative organism were already present among the animals, it might gain virulence in the wake of low-grade indigestion caused by sudden feed changes.

Herd additions purchased from stockyards sometimes initiate an outbreak. These added animals may have diarrhea at time of purchase or develop it a few days later. An outbreak so started possesses virtue in its dramatic demonstration of the folly of adding stockyards animals to clean and healthy herds.

Symptoms and Diagnosis

Udall sets the incubation period as three to seven days[1]. In a herd of about 20 cows, the following is a typical picture of the disease in a Michigan practice: Fecal material passed from two or three milk cows is observed first to change in color to a distinctive yellow. Within two hours these animals develop a definite diarrhea. Their feces retain the yellow color, become fetid, and are watery enough to splash high on a wall six feet behind the cattle.

By the following day, the diarrhea has affected all but perhaps a couple of cows in the milking line. Milk production will have dropped. It is usual for an infected herd, shipping eight cans of milk a day, to drop to four cans on the second day of the outbreak.

Veterinary help often is sought at this time. Examination discloses a picture close to the following: Rectal temperatures, except in herds where a low, moist cough is present, range from 99.5 F. to 101.5 F. Cough has accompanied the disease process in half the affected herds visited. In these, the temperatures frequently range to 103.5 F. While some affected herds maintain normal appetites, it is common to find the animals cleaning up their usual amount of chop and about half the normal hay ration. Two or three severely affected herds during a season will display nearly complete anorexia.

In more than half the herds examined, blood is present in the feces of some of the animals. Blood may be present in flecks or clots or it may make up nearly all the fecal material passed.

A recently fresh cow often suffers more severely from winter dysentery than other animals. These animals exhibit complete anorexia extending beyond cessation of diarrhea.

Rumination stops, and the cow may be unable to produce enough milk for her calf.

As a rule, cows in the milking line are the first affected. Young cattle housed with an infected herd tend to develop symptoms three or four days after the initial case. Calves only a few weeks old may also become infected.

Winter dysentery must be differentiated from hemorrhagic septicemia and coccidiosis. In winter dysentery, the temperature is lower, attitudes and appetites better on the average than in "intestinal" shipping fever. Except the moist, infrequent cough, no respiratory involvement exists.

Coccidiosis in this area is not a problem. It is eliminated on the basis of existing symptoms. Effort is made to have a number of suspect fecal samples examined for coccidia each dysentery season as a check on the accuracy of our diagnoses.

Course and Mortality

In untreated cases, winter dysentery may persist in chronic form for two or more weeks. Treatment usually effects recovery within 12 to 48 hours. New cases in an infected herd may appear for a week after the first is noticed. These delayed cases occur in partially isolated animals.

Mortality rate is low even in animals passing fecal material which appears to be nothing but blood. In five years, only two deaths have been attributed to the disease. One occurred in an untreated animal and the other in one inadequately treated. Both animals had passed large amounts of blood from the rectum. Death was due to acute anemia.

Treatment

The number of therapeutic agents used for treatment of winter dysentery is nearly as large as the number of veterinarians in cattle practice. Astringents used wisely appear to be the correct approach. Udall recommends 4 to 8 dr. of creolin[1].

In the writer's practice, the following therapeutic procedures have become nearly routine:

1. Herd is thoroughly examined and diagnosis made.

2. If half the milking herd is affected with diarrhea, all cows in milking line are included for treatment.

3. Three fluidounces rumeria compound® in 2 or 3 qt. of lukewarm water are given by means of stomach tube used with a Frick speculum and an injection pump.

4. Enough 60 gr. phenolsulfonate tablets are left with the farmer so that each animal under treatment receives the following dose: Beginning one hour after administration of rumeria: one tablet hourly for three doses; then four to six tablets daily as needed in divided doses.

5. Additional phenolsulfonate tablets are left for treatment of cases occurring later in young stock. Dosage here is the same as in (4) above.

6. Since a few farmers will take all water away from a "scoured" animal, caution is emphasized that a good supply of fresh water be accessible.

7. The occasional fresh cow which refuses to eat after cessation of diarrhea must be treated symptomatically with rumen stimulants in large amounts of water, tonics and intravenous dextrose. Such a cow is usually the only reason a veterinarian must make a return visit to a herd infected with winter dysentery.

8. The accompanying cough disappears without special treatment.

9. Owners of infected herds are cautioned to stay out of neighbors' barns and to warn visitors away from their stables during the course of the disease.

Bacterins have been used in treating winter dysentery. Their value here may be questioned from two standpoints: First, death or recovery would have been effected by nature in the time needed for such immunizing agent to be effective. Second, it may be a Vibrio that is the cause of the disease, and bacterins prepared from organisms isolated from cattle may not contain the causative microorganism.

Personal Clean-Up

Proper cleansing of equipment and person after treatment of winter dysentery does more than fulfill the veterinarian's obligation to avoid spread of disease. It leads his client to remark "Doc sure as heck won't spread those germs anywhere else." That's high-priced, ethical advertising—for free.

Effective cleansing and disinfection can be fast and simple. Frick speculum, stomach tube, injection pump, pail, boots, hands are washed in a solution of household detergent. After washing, all items are rinsed in a solution of quaternary ammonium compound or other disinfectant of choice. Coveralls are discarded until laundered. This type of cleansing and disinfecting has been used over a five-year period. During this time, no indication has appeared that the causative agent of winter dysentery has ridden to susceptible herds on the writer's person or equipment.

®Pitman-Moore Company, Indianapolis, Ind.

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Benzene Hexachloride Poisoning Of Emaciated Sheep

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THE use of benzene hexachloride (properly hexachlorocyclohexane, abbreviated BHC) as a parasiticide is well established. During 1951, the use was extended to include official dipping for scabies control in Texas and in some other states. While almost all other recommendations for the use of BHC have been for 0.025 to 0.05% of gamma isomer in the finished dip, recommendations for mange control, including sheep scabies, have been or approximated 0.06% of the gamma isomer.

Radeleff and Bushland[6] have pointed out that the gamma isomer of benzene hexachloride if applied in excessive amounts is toxic to most farm animals. In small scale tests by Bushland, Wells, and Radeleff[1] sheep were not particularly susceptible. Wethers and nonlactating ewes suffered no apparent harm from being dipped in 0.15 to 0.18% gamma isomer of BHC eight times at four-day intervals. Other mature sheep appeared unharmed by a single dipping in solutions containing 0.25% gamma isomer. Kemper and Roberts[4,5] dipped both shorn and unshorn sheep, using concentrations ranging from 0.0075 to 0.06% of gamma isomer of BHC and reported no harmful effects. Dicke,[3] in trials at the Wisconsin experiment station, used 1% gamma isomer powders and reported no harmful effects.

Several million sheep have been dipped in suspensions containing 0.06% gamma isomer of BHC; some 1,700,000 having been so dipped under supervision of Texas Livestock Sanitary Commission employees up to May 1952[2]. There had been no reported losses other than those considered normally incident to dipping operations.

At Brady, Tex., on April 30, 1952, approximately 50 ewes and 40 lambs were dipped under Livestock Sanitary Commission supervision in a suspension containing 0.06% of gamma isomer. A significant proportion of that group died of symptoms suggestive of benzene hexachloride poisoning.

The following week a flock of scabies-infested sheep was similarly dipped. This flock consisted of recently shorn ewes and rams and of suckling lambs. The same vat was used but it had been carefully cleaned and recharged just before dipping. This preparation was done by the inspector in charge of scabies control in Texas and was witnessed by several other persons. Fifty pounds of a BHC wettable powder containing 10% gamma and 60% other isomers was pre-slurried and added to the 990 gallons of fresh water in the vat. An electric pump continuously picked up dip from the bottom of the vat and returned it along the dip surface.

The sheep were dipped between 10:30 a.m. and 4:30 p.m. on a very warm day, May 7, 1952. At 5:00 p.m. some of the first animals treated were showing symptoms of poisoning. The number affected increased until darkness prevented further observation. On May 8, there were several dead and more became affected. Practicing veterinarians of Brady examined these sheep and gave a probable diagnosis of insecticide poisoning. Two affected ewes, one dead ewe, and one affected lamb were sent on their suggestion to the United States Department of Agriculture laboratory at Kerrville. These sheep exhibited typical symptoms and lesions of chlorinated hydrocarbon insecticide poisoning[1,6,7,8].

On May 9, the authors went to Brady at the request of the Texas Livestock Sanitary Commission. Observations are summarized in table 1. More sheep had been affected, according to local observers, than is reflected in the table. However, the data are reasonably descriptive of the condition of the flock 48 hours after treatment.

The affected sheep observed displayed typical symptoms of chlorinated hydrocarbon insecticide poisoning. Fifty sheep were subjected to autopsy and the lesions found were of the nature expected in such poisoning. Clinically, it was obvious that the sheep were all emaciated, with the exception of the rams. The 50 autopsies revealed not a gram of fat. In all cases the normal depots had been replaced by

*Bureau of Animal Industry, **Bureau of Entomology and Plant Quarantine, Agricultural Research Administration, U. S. Department of Agriculture.

a gelatinous orange to red-colored material.

As shown in table 1, the rams (in good condition) were unaffected and the ewes not lactating were moderately affected, but the lactating ewes were the most severely harmed. The nursing lambs were not more affected than their dams.

TABLE 1. Status of Poisoned Flock of Sheep at Brady, Tex., 48 Hours after Dipping in 0.06% Gamma Benzene Hexachloride for Scabies Eradication

	Dead	Sick	Apparently Unaffected
Rams	0	0	39
Ewes, nonlactating	12	0	352
Ewes, lactating	107	26	192
Lambs, nursing	14	115	168

Our analysis of the distribution of losses, condition of the sheep, and the circumstances surrounding the dipping was that the emaciation of the sheep was the primary cause of the losses — admitting, meanwhile, that a very slight possibility existed that the formulation might have been at fault.

Because emaciation appeared to be the predisposing factor in these poisonings and emaciated sheep can be found in many regions each year, sheep ranges being frequently subject to drouth, the present series of experiments was considered to be urgently needed to prevent further losses.

Objectives

This study had the following objectives:

1. To determine the relative toxicity of BHC to emaciated sheep and sheep in good condition.
2. To compare the toxicity of at least three different formulations of BHC from as many different manufacturers.
3. To determine whether the susceptibility, if it existed, was due to local conditions of nutrition or would apply to other emaciated sheep from other localities.
4. To evaluate the presence of intestinal parasites as a factor and determine the relation of blood picture to toxicity.
5. To test the possibility of a faulty formulation in the original dipping.
6. To determine whether the increased susceptibility to BHC poisoning, if existent, extended also to toxaphene and chlordane.

Experimental Animals

Sheep were purchased in the Brady area from a ranch ¼ mile from the source of the sheep killed by the Brady dipping. These animals were in a similarly emaciated condition (fig. 1), were recently shorn, and were of the same general conformation. In the body of this report these sheep will be referred to as "Brady sheep."

A second flock of recently shorn sheep was purchased from a ranch near Sonora, Tex., about 100 miles from Brady. This group was somewhat less emaciated than that from Brady. This group is designated "Sonora sheep."

A third lot, recently shorn, was selected from the United States Department of Agriculture flock at Camp Stanley, Tex. These were in excellent condition, having been well cared for during the winter. These will be referred to as "Stanley sheep."

All ewes had suckling lambs. In each lot, the ewes were matched with their lambs and divided into groups of ten ewes and lambs or five ewes and lambs, as the experiments required.

The Brady sheep were kept on a low maintenance diet of poor quality hay until treated, then they were fed all the alfalfa hay they desired. The Sonora sheep were dipped the day after delivery to the laboratory, then fed alfalfa hay. The Stanley sheep were kept on grass pasture until treated, then fed alfalfa hay.

Fig. 1. Two ewes representative of the Brady sheep classified as "more emaciated."

Experimental Materials

Three commercial wettable powders of benzene hexachloride were used. One, from the same production lot as used by the Texas Livestock Sanitary Commission at Brady, was composed of 10% gamma isomer, 60% other isomers, and 30% inerts. This will be designated as "14% gamma technical BHC." A second formulation was composed of 6% gamma isomer, 44% other isomers, and 50% inerts. This will be designated as "12% gamma isomer technical BHC" and is one of the materials used in previous trials at the Kerrville laboratory. A third material was composed of 25% gamma isomer from lindane

and 75% inerts and will be referred to as "lindane."

For the toxaphene and chlordane studies, laboratory formulations were used consisting of 50% insecticide, 40% deodorized kerosene, and 10% emulsifier (three parts triton X-155†, one part skil 181-A††).

All dilutions were made on a weight/weight basis. Dips were mixed just before use and disposed of immediately thereafter.

Fig. 2. The most emaciated of the Brady ewes.

Procedure

In setting up the experiments, the Brady sheep were first divided into two groups of general condition, 40 pairs of ewes and lambs being somewhat less emaciated than the remainder of the flock. These groups are designated "more emaciated" and "less emaciated" Brady sheep. This division was to give some information as to the effect of condition within the flock.

These two groups and the flocks from Sonora and Camp Stanley were then subdivided into groups of ten ewes and ten lambs or five ewes and five lambs, as required.

Limited blood studies were made of 15 of the more emaciated Brady ewes before they were dipped.

All dipping was done beginning at about 10:00 a.m. and ending at various times, usually before 12:00 noon and never later than 2:00 p.m. All treatments were applied during the last two weeks in May under similar weather conditions (cool nights and warm, partly cloudy to sunny days).

Dips were prepared and used in a portable vat and were either 30 or 50 gal. per treatment. Each sheep was kept in the dip 30 seconds, drained momentarily over the vat, and then allowed to complete draining on a concrete floor. When draining was completed, the sheep were put in pens having a minimum of shade and kept there for the duration of the observation period. Care was taken to keep the sheep from drinking or inhaling the dip.

TABLE 2. Results of Dipping the More Emaciated Brady Ewes and Their Lambs in 0.06% Gamma Isomer of Benzene Hexachloride—Kerrville

Formulation Prepared From:	Number Treated	Affected: Mildly	Severely; Recovered	Fatally	Total	Unaffected
14% gamma technical BHC	10 ewes	2	1	4	7	3
	10 lambs	0	0	0	0	10
12% gamma technical BHC	10 ewes	0	2	3	5	5
	10 lambs	0	0	1	1	9
Lindane	10 ewes	3	0	1	4	6
	10 lambs	0	0	0	0	10
Total treated	30 ewes	5	3	8	16	14
	30 lambs	0	0	1	1	29

Observation was essentially continuous for the first 72 hours after dipping except during the hours of 10:00 p.m. to 8:00 a.m.

Each sheep was paint branded according to treatment. As symptoms appeared, the sheep were given an additional brand to identify them and signify the order of appearance of symptoms within the group.

A classification of "affected" was given only after two seizures of head and body jerks had been observed. In so far as was practical, at least two observers recorded the two seizures simultaneously. Depression, as a premonitory symptom, served only to call attention to particular animals so that they were closely watched to note onset of more characteristic

TABLE 3. Results of Dipping the More Emaciated Brady Ewes and Their Lambs in 0.03% Gamma Benzene Hexachloride—Kerrville

Formulation Prepared From:	Number Treated	Affected: Mildly	Severely; Recovered	Fatally	Total	Unaffected
14% gamma technical BHC	10 ewes	1	1	0	2	8
	10 lambs	0	0	0	0	10
12% gamma technical BHC	10 ewes	2	0	0	2	8
	10 lambs	0	0	0	0	10
Lindane	10 ewes	0	1	0	1	9
	10 lambs	0	0	0	0	10
Total treated	30 ewes	3	2	0	5	25
	30 lambs	0	0	0	0	30

†Manufactured by Rohm & Haas Company, Philadelphia, Pa.

††Manufactured by Gallowhur Chemical Corporation, New York, N. Y.

symptoms. Sheep were not designated as "severely affected" until at least one violent convulsion had been observed.

Experimental Results

Benzene Hexachloride. Results of dipping sheep in 0.06% gamma BHC are given in tables 2, 4, and 5; of dipping in 0.03% gamma in table 3; and of dipping in 0.25% gamma in table 6.

These experiments resulted in unmistakable poisoning of the dipped sheep. Typical symptoms and lesions were observed and were identical with those observed at Brady on May 9.

Onset of symptoms was as early as five hours after dipping and as late as 30 hours. Deaths occurred in from 18 to 48 hours after dipping. In each series the sheep dipped in the 14% gamma material were the first to be affected. This was considered to be due to the highly irritant properties of the material. All persons contacting this product suffered severe irritation. The hands and arms of the

TABLE 4. Results of Dipping the Less Emaciated Brady Ewes and Their Lambs in 0.06% Gamma Benzene Hexachloride—Kerrville

Formulation Prepared From:	Number Treated	Affected: Mildly	Severely; Recovered	Fatally	Total	Unaffected
14% gamma technical BHC	10 ewes	0	3	1	4	6
	10 lambs	0	0	0	0	10
12% gamma technical BHC	10 ewes	0	0	0	0	10
	10 lambs	0	0	0	0	10
Lindane	10 ewes	0	0	0	0	10
	10 lambs	0	0	0	0	10
Total treated	30 ewes	0	3	1	4	26
	30 lambs	0	0	0	0	30

workers holding the sheep in the dip became inflamed and very red in color, the effect being intensified in direct sunlight. The sheep dipped in that material gave evidence of similar irritation. It may be that the increased circulation of blood in the skin due to the vasodilation caused a more rapid absorption.

Although the 14% gamma material affected the sheep most rapidly, all three formulations caused poisoning. Within treatment groups the differences between formulations were not great, but a summation of the experiments (table 7) shows that considerably more sheep were affected by 14% gamma BHC than by the other mixed isomer product or by lindane.

The more emaciated Brady ewes were affected not only by the 0.06% dips (table 2), but also by dips containing 0.03% gamma isomer (table 3). The Sonora sheep at 0.06% (table 5) and those Brady sheep at 0.03% (table 3) exhibited similar reactions. The

TABLE 5. Results of Dipping Emaciated Sonora Ewes and Their Lambs in 0.06% Gamma Benzene Hexachloride—Kerrville

Formulation Prepared From:	Number Treated	Affected: Mildly	Severely; Recovered	Fatally	Total	Unaffected
14% gamma technical BHC	10 ewes	1	1	1	3	7
	10 lambs	0	0	0	0	10
12% gamma technical BHC	10 ewes	0	0	0	0	10
	10 lambs	0	0	0	0	10
Lindane	10 ewes	0	0	0	0	10
	10 lambs	0	0	0	0	10
Total treated	30 ewes	1	1	1	3	27
	30 lambs	0	0	0	0	30

Sonora sheep were, as has been pointed out, in better condition than the thinner Brady sheep. The less emaciated of the Brady sheep (table 4) reacted about like the Sonora sheep (table 5) to 0.06% of gamma isomer.

The reactions of the ewes in good condition dipped in 0.25% of gamma BHC (table 6) are comparable to those shown by the more emaciated ewes dipped in 0.03% (table 3) and the less emaciated ewes in 0.06% gamma BHC (tables 4 and 5).

TABLE 6. Results of Dipping Well Fed Ewes (Stanley Sheep) and Their Lambs in 0.25% Gamma Benzene Hexachloride—Kerrville

Formulation Prepared From:	Number Treated	Affected: Mildly	Severely; Recovered	Fatally	Total	Unaffected
14% gamma technical BHC	10 ewes	3	0	0	3	7
	10 lambs	0	0	0	0	10
12% gamma technical BHC	10 ewes	1	0	0	1	9
	10 lambs	0	0	0	0	10
Lindane	10 ewes	0	2	0	2	8
	10 lambs	0	1	0	1	9
Total treated	30 ewes	4	2	0	6	24
	30 lambs	0	1	0	1	29

Since both the lindane preparation and the two preparations made from technical BHC caused poisoning, it seems that any dip containing 0.06% of the gamma isomer might poison similarly emaciated sheep.

Of particular note is the fact that in all

these tests the ewes were considerably more susceptible to poisoning than were their nursing lambs. There has been no previous record that would indicate that this could be expected.

No significant correlation of the simple blood picture with susceptibility could be made in this study. The results are given in table 8.

The ewes dying of poisoning were subjected to autopsy. Other than the typical lesions, the emaciation was outstanding. Not a single sheep had so much as a half gram of fat. Even the yellow bone marrow had been replaced by

TABLE 7. Summary* of Results of Dipping Emaciated Ewes and Their Lambs in 0.06% Gamma Benzene Hexachloride—Kerrville

Formulation Prepared From:	Number Treated	Affected				Unaffected
		Mildly	Severely; Recovered	Fatally	Total	
14% gamma technical BHC	30 ewes	3	4	7	14	16
	30 lambs	0	0	0	0	30
12% gamma technical BHC	30 ewes	0	2	3	5	25
	30 lambs	0	0	1	1	29
Lindane	30 ewes	3	0	1	4	26
	30 lambs	0	0	0	0	30
Total treated	90 ewes	6	6	11	23	67
	90 lambs	0	0	1	1	89

*Combining tables 2, 4, and 5 to make this table.

a gelatinous material. The normal epicardial, perirenal, omental, and mesenteric deposits were absent or replaced by gelatinous material.

A careful check of internal parasites was made. No significant infestations were found. Occasional *Haemonchus contortus* and *Cysticercus tenuicollis* were found. One ewe showed approximately 50 *H. contortus* in the abomasum and this was the most found.

Toxaphene and Chlordane. Results of dipping the more emaciated Brady sheep in 0.5

TABLE 8. Comparison of Toxic Reaction to Simple Blood Picture in 15 Emaciated Brady Ewes Dipped in 0.06% Gamma Benzene Hexachloride

Reaction to Dipping	Number of Sheep	Hemoglobin gm./100 ml.	Red Cells, Millions per cmm.	Leukocytes, Thousands per cmm.	Packed Red Cell Volume, %
Unaffected	6	14.3	8.82	6.31	40.7
Affected, recovered	4	16.2	9.22	5.60	41.2
Died	5	15.3	9.0	5.78	42.1

and 1% toxaphene and in 0.5, 1, and 2% chlordane are given in table 9. Results of dipping emaciated Sonora sheep in 1% chlordane and 1% toxaphene are given in table 10.

The 1% concentration in each case is twice that normally recommended or suggested for field use. From the results given, it is apparent that emaciated, lactating ewes are more susceptible to chlordane and toxaphene poisoning than other sheep, as, for instance, in earlier work[1] sheep withstood eight applications of 1.5% toxaphene at four-day intervals. The increase in susceptibility is not so marked, however, as it is with BHC.

Conclusions

1. Lactating, emaciated ewes are approximately eight times as susceptible to BHC poisoning as are lactating ewes in good condition.

Fig. 3. Group of more emaciated Brady ewes and lambs. One ewe dead of BHC poisoning in right foreground.

2. The normally recommended dips containing 0.03 to 0.06% gamma isomer of BHC are definitely toxic to emaciated, lactating ewes, but their lambs are more resistant.

3. Suspensions of 0.03 and 0.06% gamma isomer of BHC were toxic to emaciated, lactating ewes, whether prepared from lindane or from technical benzene hexachloride. A suspension prepared from 14% gamma technical BHC was highly irritating to the skins of workers and sheep and, perhaps, for this reason was more rapidly toxic to sheep and may have contributed to the greater number affected.

4. Lactating ewes are more susceptible to BHC poisoning than wethers or nonlactating ewes, the present data being compared with previously published data.

5. Increased susceptibility to BHC poisoning was shown in emaciated, lactating ewes from separated areas of Texas.

6. Internal parasites were present in such low numbers, or not at all, that they could not be considered as contributing factors.

7. No correlation of simple blood picture with toxicity could be made in these limited studies.

TABLE 9. Results of Dipping More Emaciated Brady Ewes and Their Lambs in Toxaphene Emulsions and in Chlordane Emulsions—Kerrville

Chemical and Concentration	Number Treated	Affected: Mildly	Severely; Recovered	Fatally	Total	Unaffected
Chlordane, 2.0%	5 ewes	0	0	1	1	4
	5 lambs	0	0	0	0	5
Chlordane, 1.0%	5 ewes	1	0	0	1	4
	5 lambs	0	0	0	0	5
Chlordane, 0.5%	10 ewes	0	0	0	0	10
	10 lambs	0	0	0	0	10
Toxaphene, 1.0%	5 ewes	0	1	0	1	4
	5 lambs	0	2	0	2	3
Toxaphene, 0.5%	10 ewes	0	0	0	0	10
	10 lambs	0	0	0	0	10

8. The increased susceptibility of emaciated, lactating ewes is found with toxaphene and chlordane but not to the extent observed with BHC.

Summary

Observations on relatively large losses of emaciated ewes and lambs in a flock of sheep dipped for scabies control suggested experiments to determine the cause of poisoning by what had previously been considered a nontoxic concentration of benzene hexachloride.

Emaciated ewes and their lambs were purchased from Texas ranches 100 miles apart and their susceptibility to gamma benzene hexachloride poisoning determined in comparison with well fed ewes and their lambs. The susceptibility of the emaciated ewes to toxaphene and chlordane poisoning was also studied.

TABLE 10. Results of Dipping Emaciated Sonora Ewes and Their Lambs in Toxaphene Emulsion and in Chlordane Emulsion—Kerrville

Chemical and Concentration	Number Treated	Affected: Mildly	Severely; Recovered	Fatally	Total	Unaffected
Chlordane, 1.0%	10 ewes	0	0	0	0	10
	10 lambs	0	0	0	0	10
Toxaphene, 1.0%	10 ewes	0	0	0	0	10
	10 lambs	0	1	0	1	9

The experiments demonstrated that lactating, emaciated ewes could be poisoned by dipping them in gamma benzene hexachloride at concentrations as low as 0.03% gamma isomer, regardless of the source of the gamma isomer (technical BHC or lindane). It was also demonstrated that lactating ewes were more susceptible to BHC poisoning than other sheep as reported in the literature, although well fed lactating ewes appeared about eight times as resistant as emaciated, lactating ewes.

No correlation of simple blood picture nor of internal parasitism could be made with the intoxications occurring.

It was also shown that emaciated ewes nursing lambs were more susceptible to toxaphene and chlordane poisoning than well fed sheep had been. Concentrations of 1% of toxaphene and chlordane injured a smaller proportion of sheep than did dips containing 0.06% gamma BHC.

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Some Observations On The Problems Of Arizona Paracolon Infections Of Poults

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SEVERAL references have appeared in veterinary literature regarding infection in turkey poults by an Arizona strain of paracolon, Ar. 7:1,7,8. Since the poultry pathology section of the San Diego Livestock Department has been dealing with this group of organisms for some years, some observations recorded in this laboratory may be of interest.

The typical ranch history of this disease is as follows: Losses, varying from 0.5 to 50% may occur from the fourth to fifth day through the third week in untreated flocks. Birds become dull and listless, and sit on their hocks for several days before death. Leg paralysis and blindness in one or both eyes is common. Pasting of the vent is sometimes observed.

On autopsy there is almost invariably demonstrated retained yolk, swollen and congested livers, and sometimes caseous plugs in the ceca. Erosion of the gizzard is seen occasionally.

Bacteriological cultures of the heart, liver, spleen, and ceca are routinely performed. Any organism fermenting dextrose, maltose, and mannite with gas in 24 hours, but not lactose, sucrose, or dulcitol, and which is motile and negative for indol is regarded as suspicious. The culture is then tested serologically with a polyvalent antiserum and a specific, Ar. 7:1,7,8, antiserum obtained from Doctor Edwards of the U. S. Public Health Service in Atlanta, Ga. Arizona paracolon organisms frequently have been isolated concomitantly with *Escherichia coli,* Pseudomonas sp., and Proteus sp. or other paracolons.

For the past year unsuccessful effort has been made to find some correlation between outbreaks of this disease and the breeder flocks. Egg shipments from the same breeder flock at the same time have been traced to several ranches only to find that one ranch suffered mortality while the others did not. Eggs from a suspected flock, with known history of paracolon infection, have been checked throughout an entire season without finding outbreaks.

A survey of the entire egg production season would seem to indicate that poults hatched early and late in the season are more susceptible to the disease than those hatched in the peak period.

An attempt was made to isolate the organism from a flock of 200 hens having a history of a severe mortality (50%) from the disease when they were poults. The vents of these birds were swabbed with a sterile swab, especially constructed for the purpose, and these specimens placed immediately into tubes containing selenite enrichment media. Subcultures of this growth were plated on desoxycholate agar. The results were completely negative.

During 1951, in conjunction with a testing program for *Salmonella typhimurium* a total of 204 breeding birds were autopsied, and all organs routinely cultured for the entire group. No cases of Arizona paracolon were isolated.

A survey of the wild life in the area of turkey ranches, revealed that the organism often can be isolated from snakes, and occasionally from gophers. This supports observations previously made by Hinshaw and McNeil[4].

An experiment was set up last fall using 21 poults obtained from an area where Arizona paracolon has not been reported. These poults were divided into three groups of seven each. Groups 1 and 2 were fed 2 cc. of a 24-hour broth culture of an Arizona paracolon organism recovered from a flock which had suffered a 25% loss. Group 3 was kept as a control.

Groups 1 and 3 were placed in an ordinary battery brooder. Group 2 was subjected to the severest possible temperature changes, ranging from 4 C. to 40 C., for 15-minute periods, repeated frequently. They were kept on paper that was never changed so that they were constantly exposed to their own droppings. Water and feed were periodically withheld.

After 14 days one bird in group 2 died. Autopsy revealed retained yolk and swollen

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liver. Arizona paracolon was isolated from the heart and liver.

The other birds did not become sick and were autopsied at the fifth week. The cultures from these were negative.

Practically all the poults that die in this area are submitted for routine bacteriological study. On ranches where there is no loss, a few of the weaker birds are sent in for routine examination.

An analysis of these poults reveals that *E. coli* and *E. communior* cause more mortality than the paracolon group, and of the paracolons, the sucrose positive show up more frequently than the Arizona group.

There have been four cases of Arizona paracolon infection so far in 1952 in the County. Of these, only one was considered a disease problem. The other three were isolated from the routine autopsies mentioned previously.

Summary

Further evaluation is needed to determine accurately the pathogenicity and significance of Arizona paracolon. The writers' opinion is that it has been over-rated as a primary disease producer in poults.

Since a picture of septicemia without coexisting toxemia caused by retained yolk was not observed as in paratyphoid, it is believed that the Arizona paracolon should be considered an opportunist which becomes pathogenic only in the presence of some adverse management factor. Such factors also tend to pave the way for invasion of *E. coli*, Proteus sp., and Pseudomonas sp. which under normal conditions are non-pathogenic.

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Johne's Disease

(*Continued from page 47*)

numerous animals. As a rule, the origin of the disease in a given herd could be established, and conditions favoring the spread within the herd were always quite evident. In some cases, the infected animal, already well along in the progress of the disease, had degenerated into the poorer class and become part of the pernicious traffic in animals that goes from farm to stockyard, to farm, and back again to the stockyards. In some of these cases, the farm from which the diseased animal came could not be determined.

Johne's disease is known to occur in sheep in this and other countries. It is not, primarily, a disease that spreads readily when animals are on pasture, but, once introduced into a flock, it might well become a serious threat if the flock were kept in rather close confinement in the same barn and yards, even if only for short periods during the year.

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Hog Cholera Vaccine

(*Continued from page 50*)

were sows when both sows and litters were challenged at the same time.

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A Californian was issued a patent (no. 2,618,216) on a process to clean "heavy dirty" eggs. Cleaning is performed by subjecting eggs to a sand blast, washing in boiling water, and coating with sterile oil. The patent-holder claims less shrinkage and better keeping qualities for the treated eggs.

Terramycin In The Treatment Of Streptococcic Mastitis†

JOSEPH SIMON, M.S., D.V.M., Ph.D., and E. GILMORE SCHMIDT, B.S., M.S., Madison, Wisconsin

FINLAY, *et al.*,[1] first described the antibiotic terramycin which was obtained from the Actinomycete, *Streptomyces rimosus*. They found that this antibiotic had a broad spectrum of activity.

Morse, *et al.*,[2] tested *in vitro* activity of this antibiotic against a number of pathogenic bacteria isolated from animals and demonstrated the broad spectrum of bacteriostatic activity of this antibiotic against animal pathogens.

Cromley and Hagely,[3] described some clinical studies on the effectiveness of terramycin in small and large animals, including two clinical trials in which the antibiotic was used against staphylococcic mastitis.

The present study was undertaken in an effort to determine the therapeutic effectiveness of terramycin in streptococcic mastitis.

Materials and Methods

Experiment 1. Irritability Trials. Because the irritability of terramycin for the bovine mammary gland had not been established, terramycin in two vehicles was infused into the mammary gland of four experimental cows according to the following schedule:

Cow 16. Each of the four quarters was infused with 1 tube, 7.1 gm. of terramycin* ointment containing 30 mg. of terramycin hydrochloride per gm.

Cow 37. Same schedule as cow 16.

Cow OE10287. Each of the four quarters was infused with 250 mg. of terramycin hydrochloride* (intravenous) in 10 ml. of sterile glass-distilled water.

Cow 182. Same schedule as cow OE10287.

The criteria used to determine irritability consisted of the following: catalase test, chloride determination, cell counts, strip-cup test and clinical examination. Each quarter of each of the four cows was sampled twice, at an eight-day interval, prior to treatment, and every 12 hours from the time of infusion through 96 hours. The cows were free of mastitis as determined by laboratory and clinical tests.

The results of the irritability studies indicated that terramycin, either in an ointment vehicle or in an aqueous solution, does not incite an inflammatory response of the bovine mammary gland and approximates penicillin in this respect.

Field Trials

The six experimental herds used for the field trials were located in the immediate area of Madison, Wis. The herds were maintained under average dairy farm conditions and had numerous cases of mastitis. Each herd was tested by individual quarter sampling utilizing the Hotis test, direct microscopic films of incubated samples, and by streaking each incubated sample upon a blood-agar medium containing 0.5% mannitol. The infected quarters were divided on a herd basis, randomization of sampling, into two nearly equal and comparable groups so that each medicament was employed under similar conditions. The terramycin was employed in an ointment vehicle whereas the vehicle for the penicillin was penicle®, a water-in-oil emulsion type of vehicle. Following treatment, each infected quarter was sampled weekly for three consecutive weeks by means of the same procedures which were employed in determining the original infectivity. A quarter was considered free of streptococci if it was negative to each of the consecutive post-treatment tests during the three-week period.

Experiment 2. 30 mg. of terramycin hydrochloride compared with crystalline penicillin.

Group A. 100,000 units of crystalline penicillin G, potassium salt in 10 ml. of penicle.

Group B. 7.1 gm. of terramycin ointment containing 30 mg. of terramycin hydrochloride per gm.

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*Furnished through the courtesy of Chas. Pfizer & Co., Inc., Brooklyn, N. Y.

®Wallace Laboratories, Inc., New York, N. Y.

The results of experiment 2 were as follows:

Group	No. Quarters Treated	Quarters Freed of Infection No.	%
A	61	46	75.4
B	65	38	60.0

The statistical evaluation of experiment 2 indicates that 100,000 units of crystalline penicillin G, potassium salt in 10 ml. of penicle is a more effective therapeutic agent for mastitis caused by *Str. agalactiae* than 7.1 gm. of terramycin ointment containing 30 mg. of terramycin hydrochloride per gm. (Chi-square= 4.07; .05> P> .025 where P is the probability.)**

Experiment 3. 60 mg. of terramycin compared with crystalline penicillin.

Group A. 100,000 units of crystalline penicillin G, potassium salt in 10 ml. of penicle.

Group B. 7.1 gm. of terramycin ointment containing 60 mg. of terramycin hydrochloride per gm.

The results of experiment 3 were as follows:

Group	No. Quarters Treated	Quarters Freed of Infection No.	%
A	69	39	56.5
B	69	41	59.4

The statistical evaluation of experiment 3 indicates that 7.1 gm. of terramycin ointment containing 60 mg. of terramycin hydrochloride per gm. is as effective a therapeutic agent for mastitis caused by *Str. agalactiae* as 100,000 units of crystalline penicillin G, potassium salt in 10 ml. of penicle, water-in-oil emulsion. (Chi-square value=.119; .50< P< .75 where P is the probability.)**

Discussion

The irritability studies of terramycin administered as an ointment or in a water vehicle indicated that this antibiotic is well tolerated by the mammary gland.

In experiment 2 the concentration of terramycin hydrochloride at 30 mg. per gm. was not of sufficient quantity and was inferior to 100,000 units of penicillin.

In experiment 3, a higher concentration of terramycin hydrochloride, 60 mg. per gm., was as effective a therapeutic agent for *Str. agalactiae* mastitis as a single treatment with 100,000 units of crystalline penicillin G, potassium salt, in 10 ml. of penicle.

**The statistical aid of Dr. Robert G. D. Steel, station statistician is gratefully acknowledged.

The curative percentage of penicillin was somewhat lower in experiment 3 than in experiment 2. This reduction in efficacy of treatment is believed to be the result of reduced sanitary efforts because of the harvest season.

Summary

1. Terramycin hydrochloride, administered either in an ointment vehicle or in an aqueous solution was well tolerated by the mammary gland.

2. Terramycin ointment, 7.1 gm. containing 30 mg. of terramycin hydrochloride per gm., was inferior to 100,000 units of penicillin in the treatment of mastitis caused by *Str. agalactiae.*

3. Terramycin ointment, 7.1 gm. containing 60 mg. of terramycin hydrochloride per gm., was as good therapeutically as 100,000 units of penicillin in the treatment of mastitis caused by *Str. agalactiae.*

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Uterine Movement Essential for Conception

By means of a thin rubber bag containing water, placed in the uterus of a cow in estrum, to which is connected a recording instrument, research workers have noted peristaltic-like movements of the uterus. These occur to such intensity as to be recorded when a bull is led alongside the cow, and may last for several minutes. Comparable movements are elicited shortly after the introduction of instilled semen into the uterus. An important observation is that external noises or excitement influence these rhythmic uterine contractions. They may cease entirely. In a measure, this may explain why cows fail to settle when in strange surroundings, after rough handling, or when there are strange noises, etc. In other words, mental and physical equilibrium are essential to conception.

Vibriosis, Its Pathogenesis And Management*

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BULLETIN 281 of the Storrs Agricultural Experiment Station entitled, Vibriosis in Cattle, reviews available information concerning the subject through December 1951. The purpose of this paper is to summarize information on the clinical manifestations, some modes of transmission, and the possible measures that may be employed in the control of this disease.

Pathogenesis

Ten herds have been blood-tested at intervals of three to six months over periods of from three to seven years. A comparison of the results of the blood tests with breeding records indicates the effect of vibriosis on the breeding efficiency of the herds, as a whole, and on individual animals.

Herd. In recently infected herds, the spread of infection was rapid in all age groups (animals over two years of age) for a period of 12 to 24 months. At some time during this period the majority of adult females reacted positively to the blood test, and from 10 to 20% aborted. Thereafter, the incidence of blood-test positive animals usually dropped to below 25%, due mainly to animals changing from a positive to a negative blood test reaction and to a lesser extent to the sale of reactors, (usually aborters or non-breeders). Routine sale of reactors is not recommended. After the period of rapid spread, infection tended to persist among the one- to three-year-old heifers. At this stage, detected abortions were not common. However, heifers that were positive at the time of service frequently required three, or more services, had undetected abortions, or were sold as non-breeders.

In the average *Vibrio fetus* infected herd, the sale of cows as non-breeders, and the number of animals requiring three, or more services, imposed a greater strain on the economy of the herd than detected abortion.

Cows and Heifers. A comparison of blood test reactions with the breeding record of individual animals showed that; (1) about 35% of cows and heifers that reacted positively at the time of service required three, or more services per calving, and (2) about 35% of those that became positive three to six months after conception, aborted. These observations suggest that the pathogenic action of *V. fetus* is limited to embryonic and placental tissue. The percentage of undetected abortions manifested only in the form of skipped heat periods is impossible to catalog.

Bulls. No lesions attributable to *V. fetus* have been observed in bulls. Observations on diluted bull semen containing added *V. fetus* cells indicated that the organism has little, if any, effect on spermatozoa. The importance of infection in bulls, appears to be concerned with the spread of the disease.

Transmission

Available information indicates that infection may be spread by service to an infected bull. Contact transmission from female to female has also been demonstrated but the actual mode of this type can only be left for speculation on the basis of present knowledge. Vulva to vulva transmission seems most logical at the time of this writing.

Artificial insemination can contribute to the spread of vibriosis, or if properly employed, can be a prime aid in its control. In transmission experiments conducted at Storrs, infection was established in heifers by artificial insemination, with diluted semen to which *V. fetus* organisms had been added.

Management

The following list of suggestions is worthy of the careful consideration of any practitioner engaged in sterility work with cattle where vibriosis may be a problem:

1. Use laboratory tests (bacteriological plus blood and cervico-vaginal mucus serology) to aid in the diagnosis of vibriosis in herds where breeding records (abortion, skipped heat and repeat breedings) suggest the presence of the disease. A reasonable diagnosis is essential to sound management of any disease problem. Routine herd tests are not recommended. Routinely, once a herd diagnosis is made all

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animals meeting the clinical requirements should be handled as if infected.

2. Blood-test cows and bulls for addition to herds at time of purchase, and again one month later, keeping animals isolated during this period. Preferably, buy negative animals or with the provision that they be found negative when tested following arrival.

3. Isolate every cow at calving time so that uterine excretions will not become widely disseminated. Clean stall and disinfect thoroughly before using for any other animal.

4. In infected or even noninfected herds, breed artificially using diluted semen containing 500 to 1,000 micrograms of dihydrostreptomycin sulfate per milliliter. Treated semen should stand three to four hours before using. Bulls negative to blood test are of course preferred. Where cooperative association semen is used, the chances are good that it is already treated. The technic is not so difficult that it cannot be employed within a herd if desired.

5. Treat heifers or cows that fail to conceive on two to three services and which show no abnormality on rectal examination with an intrauterine infusion of 1 gm. of streptomycin in 20 ml. of sterile water. Treatment at any stage of the heat cycle is safe and effective with streptomycin. It should be stated here that a rest period of two or three months without service will also often effect a clinical recovery. However, good reproductive and productive efficiency may be maintained by streptomycin therapy at negligible cost. Aureomycin and terramycin have also been employed with good results. Occasionally, aureomycin has proved irritating when administered to an animal that was not in heat. Preliminary data indicate that streptomycin intrauterine infusions at the heat prior to expected breeding as a routine practice may aid in improving reproductive efficiency in general in herds where vibriosis is a problem.

6. Treatment of heifers or cows, that have early abortions, with streptomycin in the uterus may permit quicker return to normal reproductive efficiency.

7. Routine pregnancy and sterility examination within the herd are essential to adequate control of reproductive efficiency under any circumstances. Pregnancy examination should be made ideally early enough to be an aid in the detection of early abortions.

Summary

Abortions, skipped heats (in many cases undetected abortion), and apparent delayed conception are the main clinical manifestations of vibriosis. The two latter are often more important than the first in reducing breeding efficiency and effecting the overall economy of the herd.

Artificial insemination with streptomycin-treated semen and intrauterine streptomycin treatment of problem cows and heifers are the two most important factors of seven presented which may aid in the control of vibriosis.

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Treatment of Vaginal Prolapse (Correction of Error)

There was noted in VETERINARY MEDICINE for November 1952, page 473, that "Epidural injection of 6 cc. of 90 proof alcohol has effectively controlled persistent eversion of the vagina in cattle."

Dr. C. W. Schulz, Jefferson City, Mo., author of the statement quoted, advises of the error in percentage of alcohol and further comments on the subject as follows:

"In those cases where eversion of the vagina is a sequela of calving, prolapsed uterus, or in some cases, where no apparent cause is determined, we use 10 cc. of 2% procaine hydrochloride solution injected through the first coccygeal space, epidurally. The vulva is sutured with a deep purse string suture avoiding the urethral orifice ventrally. Where pregnancy is not a complicating factor; we also administer 3 cc. to 6 cc. of posterior pituitary extract intravenously. In the great majority of cases, this method corrects the condition and recovery is the rule. However, in those that do not respond favorably; where severe straining continues and persistent eversion of the vagina is the result, we administer 70% ethyl alcohol, 6 cc. per 1,000 lb. animal, epidurally through the first coccygeal space. This has always been successful in overcoming the straining and resultant eversion. The anesthesia thus produced lasts for from three to six weeks during which time the initial condition has corrected itself. This amount of alcohol does not render the animal unable to walk; however, some swaying may be noticed. In a number of cases treated as outlined, no untoward results have occurred."

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Henry P. Davis, dog editor of *Sports Afield* magazine, editor of Dog Encyclopedia, and author of many books on canine subjects, was elected president of the National Dog Welfare Guild. The purpose of this well known nonprofit organization is to encourage appreciation and ownership of dogs.

Tonsillitis In The Dog

DEETS PICKETT, D.V.M., M.S., Kansas City, Missouri

WHEN a practitioner neglects to examine the throat, he may overlook a condition which is closely related to many common ailments in dogs.

Kirk[1] stated that many practitioners may not be aware of the presence of tonsillitis unless a practice is made of examining the throat in cases of emesis, cough, anorexia, etc. Casey[2] stated that diseased tonsils are the source of many ailments in dogs. Fleener[3] suggested that they are a source of trouble. H. C. Smith[4] wrote that acute nephritis in old dogs may be associated with chronic tonsillitis of pyogenic or streptococcic origin. He cited such infection as being related often to arthritis, non-suppurative otitis media, iritis, chorioiditis, bronchial pneumonia, and recurrent vomiting in Boston Terriers. Other authors ascribe these and many other ailments of dogs to the tonsils. Leon Herman[5] stated that suppurative nephritis is caused usually by pyogenic streptococci derived from peripheral foci such as follicular tonsillitis. He also described a case in a man in whom a cortical renal infection and perirenal abscess followed tonsillitis. Hutyra, Marek and Manninger[6] state that nephritis in dogs sometimes develops after three or four days of acute tonsillitis.

Parkinson[10] dismisses the tonsil as being of little use to the body. It seems to be accepted by him and other investigators that, as far as this organ is concerned, the lymph flow from it originates in the tonsil itself and there are no true afferent vessels.

In view of the damage done by infected tonsils, it would appear that examination of the throat should be included as an essential part of routine physical check-up. In the writer's practice, this routine examination has disclosed astonishingly large numbers of animals, approximately 16%, with chronically enlarged and infected tonsils.

Classification

For purposes of outline, tonsillitis may be classified as acute and chronic. The acute form may, of course, be separated into follicular, parenchymatous, and superficial; this distinction is probably not necessary in veterinary medicine. Follicular tonsillitis is the usual form observed and is most frequently caused by a hemolytic streptococcus. Dinsmore[7] cited Smith as having tabulated the results of cultures of 200 tonsils removed from dogs. Of these, hemolytic streptococcus was isolated from 88; *Streptococcus pyogenes* from 23. Kirk[1] said that the acute and chronic forms of this infection are generally due to the hemolytic streptococcus. Most writers seem to agree on this point.

Diagnosis of either form is not difficult. One should familiarize oneself with the appearance of normal tonsils, which can be done by routine throat examinations. In a short time, one becomes familiar with the appearance of tonsils in varying degrees of health and disease. A healthy organ has a pink color resembling that of the surrounding mucous membrane, while one which is diseased may vary in color from light red to a dark, liver-like purple with yellowish splotches.

Acute Tonsillitis

In mild cases of acute tonsillitis, there is redness and swelling. The more severe exhibit extensive swelling and congestion and are covered usually with a thin grayish exudate or white frothy mucus. Frequently, the light

touch of a tongue depressor to an inflamed tonsil will cause bleeding. The mandibular lymph nodes are swollen and the animal may or may not exhibit pain when the mouth is opened. Respiratory infections, with dyspnea, coughing, and sneezing may be accompanying symptoms. Diarrhea is a frequent complication. Chilling may occur and there may be evidence of general toxemia. Swallowing is painful; to such extent that the animal may refuse to eat. Ptyalism is frequent, the saliva being thick and tenacious. Conjunctivitis usually is present with serous or seromucous discharge. Occasionally unilateral tonsillitis is seen. Such a case was observed in a 2½-year-old Pomeranian which was presented to the hospital with a history of one eye discharging for about five or six days. No other symptoms had been noticed by the owner. Examination revealed swelling and inflammation of one tonsil.

Primary acute tonsillitis should be differentiated from those secondary cases caused by systemic infections such as distemper and leptospirosis. The tonsils, during a siege of distemper, are almost invariably inflamed; however, they are usually only moderately swollen and superficially injected. Leptospirosis is differentiated by its more profound symptoms, rapid course, rapid dehydration, extreme soreness, precipitate drop in temperature, and orange injected sclera. Another differentiating symptom is icterus which occurs in some patients. Although Kirk,[1] and Hutyra, Marek and Manninger[8] stated that nephritis occasionally accompanies acute tonsillitis, the writer has seen little extensive kidney damage in that condition. Extensive kidney damage practically always is present in leptospirosis, as revealed by evidence of albuminuria and casts.

Infectious canine hepatitis is another disease which should be differentiated from acute tonsillitis. According to Coffin,[3] the tonsillar change in infectious hepatitis is only a minor part of the entire pathologic picture; the tonsils reflecting the generalized lymphoid reaction. There is a marked involvement of the liver which is not present in primary acute tonsillitis.

Kirk[1] reported that tonsillitis can be a severe infection, sometimes going through a kennel almost with the rapidity of a virus disease. The writer has not seen such a condition in Kansas City. One may see cases of primary acute tonsillitis in a household or one in a group of three or four dogs or even in a kennel of many dogs, as a sporadic, seemingly noncontagious entity.

Treatment

There are various methods listed in medical literature for the treatment of the acute form of this disease. These include painting the tonsils with various antiseptic preparations. This method has never greatly appealed to the writer because the major part of the infection is below the surface where it is not reached at all by topical application of a therapeutic agent. Local treatment has been employed but in no instance was inflammation relieved in this manner. Beckman[2] questioned the value of local applications. He also made the statement that according to the report of another investigator who had treated 100 people with sulfadiazine, the sulfonamides have not been successful in the treatment of streptococcal sore throat. This did not agree with the findings of Kirk[10]. According to this authority, sulfanilamide is useful in hemolytic streptococcal infections but penicillin is probably superior. Both writers agreed that penicillin is the superior agent. The writer has found terramycin, as well as penicillin to be satisfactory therapeutic agents.

The supporting treatment is as important in the therapy of acute tonsillitis as it is in various systemic diseases. This consists of intravenous fluids, vitamin therapy, intramuscular injections of liver extracts, occasional transfusions, and various symptomatic treatments such as the control of diarrhea.

Chronic Tonsillitis

Chronic tonsillitis is hypertrophy usually caused by sepsis, cold, debility, and various systemic diseases such as distemper and leptospirosis. It has been the writer's observation that about one-half of the dogs having recovered from a severe case of distemper have a residual chronic tonsillitis. Follicular tonsillitis, too, many times does not terminate with complete recovery.

A focal infection is an area of tissue infected with pathogenic organisms capable of producing damage elsewhere in the body by their toxins or by establishing secondary foci. The chronic septic absorption from tonsils causes many serious conditions such as various cardiac difficulties, chronic conjunctivitis, arthritis, nephritis, convulsions, lassitude, dull hair coat, appearance of malnutrition, lack of vigor, general muscular stiffness, dry skin, and itching and vomiting.

Chronic tonsillitis is rather easy to diagnose. Chronically infected organs when going through a phase of acute inflammation may show the same symptoms described under

acute tonsillitis; bleeding of the tonsils, sore throat, pharyngitis, pain, headache, chills, fever, cough, vomiting, anorexia, discharge from the conjunctiva, difficult swallowing, frequent "empty" swallowing, diarrhea, frequent emesis, a fluttering noise in the throat, loss of smell, painful swelling of the mandibular lymphatic glands, and peritonsillar abscesses.

A chronic enlargement of the mandibular glands on one or both sides of the neck accompanies chronic tonsillitis. When the throat is examined, the hypertrophied tonsils may stand out as protuberant masses which almost

Fig. 1. Pediculated tonsil (L).

touch one another, sometimes actually forming a mechanical obstruction in the throat. Occasionally they may not show any difference in color from the surrounding tissues. However, in these, pus and pain are frequently demonstrated by pressure with a tongue depressor. They can vary from a strawberry-like appearance to that of liver and be studded with whitish points of necrosis, pus, and bacteria.

History is usually typical in affected dogs. The owner often gives a history of vomiting when actually the animal has only made strong efforts to clear its throat. Questioning will reveal the fact that the animal may have vomited only a white, mucus-like froth. There may be frequent sneezing over a period of time. An animal with chronic tonsillitis will occasionally develop a habit of more or less constantly licking the feet or other parts of the body. Such patients may also lick the floor or walls or such inanimate objects as table legs. Unilateral chronic tonsillitis is occasionally seen. A man with a bird dog may complain that his Pointer "just can't smell a thing." When a good bird dog apparently loses his sense of smell, the possibility of chronic tonsillitis should be considered.

Dogs are sometimes presented at the hospital with a chronic ulcer on the outside of each lower lip. This is caused by constant ptyalism. Ptyalism may be caused by infected teeth or gingivitis, but it is not at all infrequently that chronic tonsillitis is found to be the cause. Local treatment of the ulcers is of small benefit unless the cause is removed. When the tonsils are the cause, they should be removed and ulcers treated by actual cautery.

Treatment of chronic tonsillitis, except during flare-ups of acute infection, is relatively unsuccessful. It is agreed that tonsillectomy is the only effective method of relieving the condition. Smith[4] suggested the removal of tonsils to prevent the recurrence of respiratory infections and also to correct recurrent vomiting so frequently found in Boston Terriers. Fleener,[5] in a case report, told of removing the tonsils of 13 of 16 full grown racing Greyhounds. These had been presented at the hospital with a history that during the racing season several of the dogs became sick, affected with sore throats and swollen tonsils. This condition appeared to affect several of the dogs at a time and during the racing season represented an economic loss. The Greyhound owner told of the tonsils being removed from three dogs of a litter of six which raced the season while the other three had sick spells. Fleener[5] explained in his article that advocating the general practice of removing tonsils from all dogs was not intended, but he did suggest that there might be a higher percentage of these organs carrying infection than is generally realized.

Kirk[10] suggested that excision of one or both tonsils may at some time or other be contemplated. Casey[9] recommended the removal of tonsils which may appear normal provided that they are extremely tender and sore when pressure is applied at the base. Dinsmore[7] recommended the removal where repeated attacks of tonsillitis disturbed the well being of the animal. Anderes[12] believes care should be used in assessing indications for tonsillectomy, but recommends removal of tonsils when they constitute a foci of infection, may be responsible for loss of olfactory sense, or be an impediment to hearing. Wakeley,[11] discussing

chronic tonsillitis in man, made the statement that operative removal is the only satisfactory treatment. That statement may also be applied to chronic tonsillitis in dogs.

Certain writers discuss tonsillectomy as if it should be approached with apprehension. Hemorrhage is feared. Kirk[10] states that tonsillectomy is an operation attended by profuse hemorrhage and no little difficulty. Dinsmore[7] wrote that occasionally there is excessive hemorrhage. Anderes[13] says that hemorrhage is unnecessary. Casey[6] wrote that he had seen no noticeable hemorrhage following that operation. In over 300 tonsillectomies, the writer has seen excessive hemorrhage in only one case. This was caused by carelessly overlooking a bleeding point and then leaving the animal in a cage without adequate postoperative inspection. When the bleeding was discovered, it was quickly stopped.

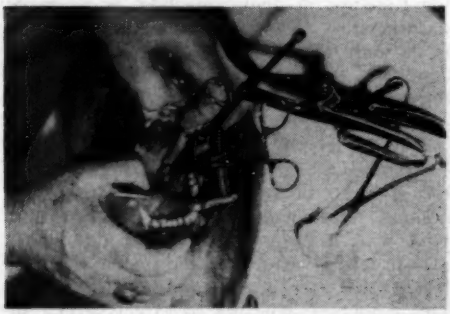

Fig. 2. Operative technic. Tonsil in loop of Tyding's snare.

When proper technic is used, a tonsillectomy may be approached with complete confidence. There are numerous methods of controlling hemorrhage. Recommendations vary with severity: application of 1:1,000 adrenalin for capillary hemorrhage, torsion of the vessels, packs applied to the tonsillar fossa, actual cautery and ligation of individual vessels, as well as other methods. The writer can recall only twice when ligation of vessels was necessary. Removal of the tonsil by a slow and steady pressure on the snare eliminates most bleeding. Vessels are closed off in this manner and many times the tonsil crypt will show no bleeding at all following this procedure.

Actual cautery with a point just hot enough to sear is the writer's favorite method of controlling hemorrhage. The cautery point should be applied to all points of bleeding or possible points of bleeding. A cautery point which is too hot should not be used because it may burn off the end of an artery thereby defeating its purpose or it may result in the deep burning of tissue which delays healing as well as causing considerable discomfort to the patient. The fossa should be carefully examined to see that no bleeders have been overlooked. When the tissues are pulled tight by forceps, these points easily can be missed. Testing should be done by relaxing the tissue held by forceps. The average tonsillectomy should not cause the loss of over 2 to 4 cc. of blood.

Surgical Technic

Normal rules applying to other veterinary surgery should apply to the preparation of a dog for tonsillectomy. Patients should be free of parasites and in as good physical condition as possible. Any acute inflammation should be allowed to subside before surgery. It has been found good practice to administer penicillin routinely for a few days prior to and following surgery. It is sometimes desirable to place the animal on a program of general conditioning with tonics, vitamins, and a nutritive diet prior to surgery. This is particularly true if there appears to be a general debility of any kind.

Fig. 3. Tonsil removed. Blood vessels touched with electric cautery point. Note dark spots in tonsillar crypt.

Dogs suffering from septic absorption but having a generally good physical condition may be operated on at any time following a short course of penicillin therapy.

Dinsmore[7] used preoperative sedation with morphine sulfate and atropine sulfate followed by pentobarbital sodium except in old and degenerated toxic patients where too great a risk would be assumed if pentobarbital sodium

were given. In these, local infiltration with 2% novocaine was described by him as being most satisfactory if preoperative sedation with morphine and atropine had been given. In other cases, where pentobarbital sodium was contraindicated, pentothal sodium was his next drug of choice; it too following morphine and atropine. The writer uses the same anesthesia methods described by Dinsmore,[7] with the exception that 1 cc. of 50 mg. demerol® per 10 lb. of body weight is administered in place of morphine. The inspections one may make of the mouth for postoperative bleeding while the animal is narcotized is one advantage of the longer lasting anesthesia obtained with pentobarbital sodium, but when it is contraindicated, pentothal sodium or local anesthesia may be used.

Some writers advise the removal of tonsils by dissection with scissors, discouraging the so-called "guillotine" or snare method. These are the writers who describe excessive hemorrhage following surgery. According to them, the complete tonsil cannot be removed with a snare. This opinion disagrees with that of many veterinarians who believe that the snare is the instrument of choice. When proper technic is used, the snare can easily remove a tonsil completely and smoothly, scissors being used only to sever the small tag of tissue which may be held in the snare following squeezing off.

The tonsil of the dog has on its medial side an accessory fold which the writer could not find described in Sisson,[13] Bradley,[14] or Miller[15]. Because this fold of tonsil tissue lies rather flat against the fossa, it is easily overlooked when the snare is applied. To avoid this, the medial fold of mucous membrane may be pulled out by forceps, thereby fully exposing this accessory tissue. Because most chronically infected tonsils are fragile and easily pulled apart, the operation is facilitated considerably if the snare* is first fitted down snugly around the tonsil, which is then gently grasped by the forceps. The tonsil may then be raised carefully and inspected, making certain that the snare is well around the base and that the entire organ is included. Pressure is then applied on the handles, pulling the snare slowly and evenly, gradually severing the tonsil from its base. A full two minutes should be taken for this process.

Examination following this will usually show a small, tough remnant of tonsillar base pulled into the end of the snare. The instrument should be slightly relaxed and gentle traction applied by the forceps, stretching this remnant of tissue which may now be severed by sharp scissors. Inspection of the tonsillar fossa usually discloses very few bleeding points. These can be rapidly controlled by cautery as described. The opposite tonsil is then removed in a like manner, after which both fossae are again examined for bleeding points. Adrenalin (1:1,000) may now be applied for control of any possible capillary bleeding. As an aid in preventing infection, powdered sulfanilamide is placed in the tonsillar fossa at this time. The animal is then placed in a warm cage where it should be inspected frequently. The above described technic has the advantages of being thorough, simple, and avoids hemorrhage.

*A Tyding's snare is preferred by the writer.
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Influence Of Protein On The Milk Fever Complex: A Four Year Study

PART III. ECLAMPSIA (PUERPERAL TETANY)

R. F. VIGUE, D.V.M., Springvale, Maine

A REVIEW of the scant literature available on the subject of eclampsia in the bitch reveals the fact that many questions regarding this metabolic disturbance are unanswered. Regarding the etiology, mention is made of theories such as uremia, absorption of toxic materials from feti and placentae, mammary toxemia, and possibly psychological disturbances such as fright, too much handling of the puppies, etc. However, no work has been undertaken to prove or disprove any of these theories. There seems to be rather general agreement that the appearance or exacerbation of the disease is etiologically connected with pregnancy and lactation. Eclampsia has also been reported in the cat by Dildine,[1] and Benesch,[2] and in the sow by Carey[3]. It has been observed by the writer in South American chinchillas.

The most apparent disturbance in eclampsia is a tonicoclonic spasm, which apparently is related to concurrent hypocalcemia. There is a reduction in the concentration of ionized calcium in the blood and generally the parenteral administration of calcium salts combined with sedation is effective therapy. It is believed that in this disease, as in milk fever of cows, the therapy is directed at a symptom or manifestation (hypocalcemia) rather than at the cause.

In a previous report on parturient paresis of the cow,[4] the writer has called attention to the fact that the amount of ionized (physiologically active) calcium is dependent upon an equilibrium between total serum calcium and the portion that is bound loosely to proteins in the blood stream, the constant for this equilibrium being altered by such factors as pH and CO_2 tension. Moreover, the extent to which calcium is bound to proteins is dependent upon the concentration of plasma proteins; and, of course, the concentration of the plasma proteins, which for the most part are synthesized in the liver, dependent upon the protein state of the animal. Thus, in essence, an acute hypocalcemia denotes an acute hypoproteinemia. In their investigations, Rawson and Sunderman[5] have shown that the portion of calcium bound to proteins can be decreased when the plasma proteins are diminished. It is probable that as long as the plasma protein level can be maintained there is no hypocalcemia, as the animal is capable of mobilizing calcium from skeletal depots, which accounts for approximately 90% of the total amount.

As indicated, this is the third of a series of discussions prepared by the author, having to do with the influence of protein on metabolic disturbances. Readers are referred to VETERINARY MEDICINE for June 1952, page 215, for part I, and December 1952, page 491, for part II. These articles refer to the influence of protein on the milk fever complex and to ketosis of dairy cattle, respectively.

Further discussion on this same subject will follow in subsequent issues of this journal.

Proteinuria Common Finding in Eclampsia

In this study of eclampsia in the bitch, proteinuria was a constant finding, being demonstrable to some extent in every instance. In a few determinations (seven), low serum protein concentrations were also recorded. The method employed by the writer was the measurement of the specific gravity of the serum in solutions of copper sulfate[6]. In those cases that terminated fatally, liver damage was prominent. This appeared to be greatest in the periphery of the lobules and consisted of hemorrhages, fatty degeneration, and toxic necrosis. In a few cases (predominantly those occurring more than a week after whelping), the renal tubules exhibited evidence of albuminous degeneration.

In an effort to clarify the etiology of eclampsia in women, numerous investigations have been undertaken to study those alterations in the normal physiology of woman brought about by pregnancy which may have some bearing upon its development. Fries and Kenny[7] found that while the total circulating protein was increased in normal pregnancy, it was reduced in severe pre-eclampsia and eclampsia, due to reduction in the plasma volume and decreased plasma protein concentration. These workers thought it significant that all of their patients with severe eclampsia gave histories of poor protein intake. Berlin and colleagues[8] studied the total red cell and

plasma volume in eight women with pre-eclampsia, by means of P^{32}-labeled red blood cells. They found an average reduction of 26% in the blood volume, 16% in the total red cell volume, and 31% in the plasma volume. MacArthur[9] studied the plasma protein levels in 600 pregnant women and observed that hypoproteinemia is common in pregnancy, that it becomes extreme in toxic patients, and then loses its normal relation to the blood volume.

In their report of 100 cases of eclampsia in women, Turner, DeLozier, and Whitacre[10] state that it was significant that a large proportion of patients received no prenatal care and concluded that this emphasized the importance of preventive measures, suggesting the possible etiological role of malnutrition. Bryant[11] and a host of others[12,13,14] have written that eclampsia in women is much less prone to develop in well nourished healthy patients on a balanced diet. In his report, Strauss[15] lays particular stress to low protein intake as a precursor; and Tompkins[16] goes so far as to postulate that eclampsia in human beings is in reality a nutritional deficiency, and asserts that in most cases of pre-eclampsia the condition is easily reversible by proper dietary treatment alone.

It is the writer's opinion that eclampsia, like its analogous conditions in other species (milk fever and ketosis in the cow and ewe, and toxemia of pregnancy in women) is perhaps related to Selye's general adaptation syndrome, the stress possibly being a nutritional (amino acids) deficiency.

In accordance with this etiological concept of eclampsia in the bitch, careful records and follow-ups were kept on 32 cases treated in the hospital. All of these bitches were bred again, some two years after the initial attack. The earliest development of the disease recorded was ten hours after whelping and the longest was four weeks; the great majority occurring within 72 hours. The 32 animals were divided into two groups; one to serve as controls, the other in which preventive measures were instituted.

Experimental

Group 1 consisted of 17 dogs varying in ages from ten months to seven years. Pregnancies were from the first to the fourth, the mean being slightly below the second. Fourteen were kennel-owned and three were family pets. At least six in this group had received calcium gluconate tablets (usually massive doses) or some other type of mineral and vitamin supplementation during pregnancy for periods varying from eight days to over one month. The failure of calcium to prevent the development of eclampsia attests that it is more than a simple hypocalcemia or calcium deficiency. These patients received the usual hospital therapy consisting of sedation, parenteral calcium, and subcutaneous injections of amino acids (parenamine®[1]). When animals were discharged, the owners were advised to feed liberal amounts of meat and eggs to prevent relapses. No recommendations were made about changing feeding practices for the next breeding. Among these clients, even if such advice had been given, it is doubtful if it would have been adhered to. In the next pregnancies, with an interval of from seven months to over two years from the last, eclampsia developed in six. This would appear to substantiate Brumley's[17] statement that recurrences are not uncommon.

Group 2 was comprised of 15 bitches in about the same age span as the other. Six were kennel-owned and nine privately owned, the reason for selecting more in the latter category being assurance that preventive measures and recommendations would be carried out. This group of dogs received the same treatment for the disease when they were presented and when the owners indicated that they would like to breed the dogs again it was emphasized that definite prophylactic measures should be taken during subsequent pregnancies. They were advised to contact the hospital soon after breeding. Although it was not specifically stated that the next heat should be skipped, the records indicate that such was the case. Only one dog was bred on the first heat; the longest interval was 29 months; the average was not quite 13 months for the group.

Preventive measures consisted in providing the pregnant bitch with an ample supply of good quality proteins such as raw horsemeat, canned fish, whole eggs, cottage cheese, and powdered skimmed milk. The better brands of the prepared (canned) dog foods were also recommended. In addition, one prenatal®[2] capsule was supplied daily to ten of the bitches for periods varying from 12 days to five weeks before the time of whelping. On the basis of statistics derived in the first group, five of the 15 bitches would have been expected to develop eclampsia. However, only one had recurrence of the disease, a kennel-owned dog whose feeding regimen was somewhat questionable.

(*Continued on page 74*)

®[1]Winthrop-Stearns, Inc., New York, N. Y.
®[2]Lederle Laboratories Division, Pearl River, N. Y.

EDITORIAL COMMENTS

Research — An Investment in Farm Progress

In a fact-giving leaflet, titled as above, and constituting a report of the University of Missouri, Division of Agricultural Sciences, Director J. H. Longwell states: "Agricultural research is an investment. Agricultural research creates new wealth, new industries, new jobs, and new products. It pays its own way many times over, not only to farmers but to the whole state community. A few examples will show that research yields monetary returns worth many times the cost. Agricultural research raises both the quantity and quality of farm products, thereby improving the living standards of all our people."

Mr. Longwell cites facts having to do with industrial research. Industrial products resulting from research and unknown 20 years ago now make up 50% of sales. In the light of this experience, no progressive industry would dare relax efforts to improve products and develop new ones.

Through research in agriculture, crop yields have increased 45% in the last 25 years. Milk production has increased 20%. The number of eggs per hen has increased 50%. Disease resistant varieties of crops have been developed and proved, better breeding of domestic livestock has made possible improved gains, and many measures have resulted in a phenomenal transformation in agricultural practices.

Agricultural research is the "life-blood" of continued improvements that will assure an adequate diet of high quality for a rapidly increasing population and a reasonable return for growers. Answers to important questions are now sought by highly trained investigators who will, with adequate support, continue to provide facts that will contribute to accumulated information so necessary to efficiency in food production.

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Recommend State Regulations Restricting Garbage Feeding

The industry front proposing legislation regulating the feeding of garbage to swine is rapidly broadening. Packing interests are also concerned. To illustrate, it is noted that a committee of the National Independent Meat Packers Association, selected to study all aspects of the problem, adopted a resolution during sessions in Kansas City, Mo., on December 12, 1952 urging vigorous efforts to eradicate vesicular exanthema and other diseases of swine by state legislative action as follows:

"1. Making it unlawful to feed uncooked commercial garbage to hogs, with appropriate penalties.

"2. Authorizing state officials to quarantine any hogs.

"3. Granting full authority to state officials to require the disinfecting of all facilities, including yards, transportation and feeding facilities, and prescribing the type of disinfecting material.

"4. Preventing the sale or movement of quarantined hogs within a state or from the state, except under proper permits.

"5. Making available appropriations which will enable the state to finance an adequate preventive and enforcement program and also enabling the state to share in the 50-50 indemnity program.

"6. Authorizing state officials to kill, bury, destroy, render, or otherwise dispose of infected hogs with proper indemnification.

"7. Authorizing state officials in their discretion to take such other preventive steps as in their judgment may be required."

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Farm Group Oppose Cooking Garbage

The California Farm Bureau Federation declared its opposition to proposed state legislation to require the cooking of all garbage fed to swine. This group recommended as an alternative, large state appropriations for research, having as the objective the development of immunizing agents and establishment of pilot plants for study of economic aspects of garbage cooking and whether such processed material is a satisfactory hog feed.

Eradication procedure will be further delayed in California if recommendations made

by this powerful group are accepted. Sufficient examples exist to demonstrate the futility of attempts to eradicate disease by vaccination.

Experimental trials have revealed the value of cooked garbage for swine feeding but even if sterilization reduced its value, the loss could hardly be reconciled with dangers that attend a compromising attitude.

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Outbreak of Foot-and-Mouth Disease on the Island of Martinique

An outbreak of foot-and-mouth disease has ocurred on the Island of Martinique, a French insular possession, one of the larger Windward Islands of the West Indies off the coast of Venezuela, South America.

The usual stringent restrictions have been imposed to prohibit importations that might serve to carry the infective virus.

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During the last year, residents of Argentina, New Zealand, and Australia ate more meat than residents of the United States.

A summary of report, based on a survey of world meat-eating trends, appearing in *The National Provisioner* for December 13, 1952, gives annual per capita consumption data in pounds as follows:

Argentina	230	United States	139
New Zealand	228	Canada	126
Australia	204	Great Britain	74

▼ ▼ ▼

A test to determine pregnancy in women depends on the shape taken by crystallizing elements of cervical mucus when smeared on a glass slide. Dr. Maxwell Roland of New York University, Bellevue Medical Center, demonstrated the simple technic before the American Academy of Obstetrics meeting in Chicago. High accuracy (99%) is claimed as early as a day or two after missed menstrual period.

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"Shipping fever" is a blanket term covering common infections of cattle, in the opinion of Dr. George R. Fowler, Iowa State College, Ames. Doctor Fowler stated at the Missouri Short Course that he believed shipping fever is due to a virus which gains its hold on the animal when it is debilitated. The uncomplicated virus infection may cause little trouble but predisposes infected animals to secondary bacterial infections which may be identified on autopsy.

Value of the Strip Cup

Writing in *Hoard's Dairyman* for December 10, 1952, Dr. I. A. Schipper states that the strip cup is the best method of mastitis detection available today. When milk is abnormal to the extent that clots will not pass through the screen or are otherwise observed, or if the milk is stringy or blood-tinged; and when these changes are associated with general depression, a swollen quarter, and elevated body temperature, the cow has mastitis. Udder changes may have been initiated by pathological processes other than infections of the mammary gland. The understanding of these causative factors is essential to an intelligent diagnosis and suggested procedure for therapy.

Emphasized in this feature article are the facts of drug fastness and danger that inadequate antibiotic therapy may lead to uncontrollable cases of the disease. Doctor Schipper states that the veterinarian should always be advised of kind, amount, and duration of any previous treatment of particular cases.

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Research Fellowship Awards Program for 1953-54

Continuing the project begun some years ago, the Ralston-Purina Company has announced their Research Fellowship Awards Program for the school year July 1, 1953-June 30, 1954. This is similar to previous annual programs.

Any individual qualified for graduate study in any land-grant agricultural college or approved veterinary school and possessing desirable personal qualifications is eligible on submission of an application. Not more than seven fellowships ($1,560. annually) will be awarded; two each for research in animal, dairy, and poultry husbandry and one in the field of veterinary science.

A select committee of five, chosen as representatives of agricultural organizations, will officiate in selection of award winners.

Application blanks for these Fellowship Awards may be obtained by writing the Awards Committee, c/o Mr. J. D. Sykes, Ralston-Purina Company, St. Louis 2, Mo.

▼ ▼ ▼

Iowa has only ten accredited herds of swine, *i.e.*, free of brucellosis infection on repeated tests.

Cash Award Contest Announced for Veterinary Students

A $3,000. cash prize contest for senior students enrolled in accredited veterinary colleges in the United States is announced by Associated Serum Producers, Inc.

Prizes will be awarded to seniors in all of the 17 colleges for the best papers written on the subject, Veterinary Public Relations. Distribution of prize money will be as follows: $1,000. national first prize; $300. national second prize; and 17 prizes of $100. each; the latter to be awarded to the winner in each of the schools.

Objective of the contest is to develop an understanding among students of some problems with which they will be confronted on entering active practice. This applies to public relations, problems of lay competition, and factors that enter into building a sound practice.

Contest Rules

Exact length of the papers to be submitted will be left to the discretion of each contestant. Background material is being sent to each senior by the Educational Committee. Each contestant will be expected to do his own research, and to develop his theme according to his own best judgment.

All papers must be submitted to Associated Serum Producers, Des Moines, Ia., by April 15, 1953. Awards will be announced, both as to individual college winners and national winners, well ahead of the end of the school year. The paper winning first award will be published in proper form and distributed nationally to members of the profession and other interested parties.

A minimum of three entrants from a college will be required for a contestant to be eligible for the cash prize designated to his college group; however, any senior can compete for the national prize, irrespective of whether his college has sufficient entries to qualify.

"We believe that this contest can be a means of helping young graduates to a much better start in practice", a spokesman for the A.S.P. group states. "Too many young men enter the profession unaware of some of the realities of everyday practice, of how to get along in a community, how to build a sound practice, and how to meet competition from unethical sources. Sound public relations for the profession are essential in all this. Associated Serum Producers has given evidence of that belief by underwriting its national public relations campaign for the profession for the last 18 years. Now, we plan to go a step further and help the graduating seniors to a better understanding of the problem. The $3,000. in cash prizes we are offering represents only a small part of the value that will be gained by seniors who enter and compete in the contest this spring."

Senior veterinary students desiring further information may write Associated Serum Producers, 830 Liberty Building, Des Moines, Iowa.

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An ordinary battery-powered otoscope, equipped with a long nose speculum, has been employed by Dr. William R. Henson, Shelby, Ohio, for the diagnosis of atrophic rhinitis in a drove of swine in his territory. A brief reference to his work appears in the January issue of the *Farm Journal.*

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Eclampsia (Puerperal Tetany)

(*Continued from page 71*)

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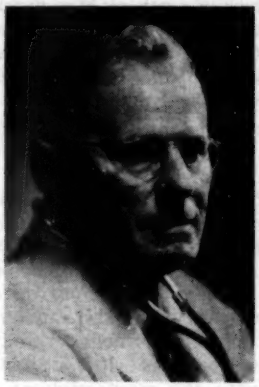
Dr. Erwin F. Schroeder
Victim of a Coronary Occlusion

Dr. Erwin F. Schroeder, former chief veterinarian of the Angell Memorial Animal Hospital, died December 26, 1952, at his ranch at Industry, Tex. He had been retired from his former position since 1950, and had been ranching at the home at which he was born in 1886.

Doctor Schroeder attended the public schools in Industry before going to the Agri-



Dr. Erwin F. Schroeder

cultural and Mechanical College of Texas, where he received a B.S. in Animal Husbandry in 1905. After spending three years on the Clifton ranch, near Waco, Tex., he matriculated in the College of Veterinary Medicine, Ohio State University and was graduated with the class of 1911.

He then came to Boston, where he became associated successively with the late Dr. Mark Simpson and the late Dr. Daniel D. Lee before going into practice in Somerville, Mass. While in practice there, he joined the visiting staff of the Angell Memorial. He gave up practice in 1917 to serve in the Army as a second lieutenant in the Remount Service. After the war, he entered private practice in Harrisburg, Ohio, but after four years, joined the Angell Memorial staff again, in 1924.

Through the medium of his many contributions to the literature on fracture treatment Doctor Schroeder became known to countless veterinarians throughout the United States. He is perhaps best known for his modification of the Thomas splint, called the "Schroeder" splint by resolution of the Massachusetts Veterinary Association. As the inventor of many mechanical gadgets used in fracture repair, Doctor Schroeder persistently minimized the importance of the gadget and stressed the necessity of increased knowledge of anatomical, functional and physical factors involved in fracture reduction and fixation.

At lectures and demonstrations before orthopedic surgeons, as well as before numerous veterinary groups, he gathered hosts of friends and admirers in both the medical and veterinary professions. He was a member and past-president of the Massachusetts Veterinary Association; a member of the New England Veterinary Medical Association, the A.A.H.A. and the A.V.M.A.

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Necrophorus Infection of the Udder

The foul-smelling infection that occurs between the anterior quarters of the udder in dairy cows is observed quite frequently in our practice. It has also been the subject of some discussion among Missouri practitioners.

For many years our experience indicated that the infection was remarkably resistant to all topical treatment.

Because of the distinctive odor that suggested necrophorus infection, it was concluded that the same organism was involved and that it differed little, except in location, from common foot rot. For some time now, all cases have been treated by the intravenous injection of 60 gm. sodium sulfapyridine in 500 cc. of distilled water. Results have been excellent with this simple treatment which is now routine in our practice.—*C. W. Schulz, D.V.M.*

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Importation of Mexican Cattle

Since the lifting of the quarantine on importation of Mexican cattle on September 1, 1952 and up to January 1, 1953, approximately 15,000 head of live Mexican cattle have been admitted through the entry point at Douglas, Ariz. This information has been supplied by Dr. F. F. Schmidt of that city. Further, we are advised that the quality of these cattle has been excellent. Most of them are dehorned and weigh around 700 lb. per head. It is expected that further importations may include lighter weights.

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Thorough disinfection and attention to other sanitary practices have made possible restocking of some broiler plants in which disastrous losses occurred due to air sac infection.

PUBLIC HEALTH

JAMES H. STEELE, D.V.M., M.P.H.
Atlanta, Georgia

Rocky Mountain Spotted Fever

Rocky Mountain spotted fever occurs during the tick season. In western states, *Dermacentor andersoni*, the sheep tick which is found on many animals, and in the east *Dermacentor variabilis*, the common dog tick, are vectors. The role of sheep and dog in maintaining the disease has been studied by Badger. He demonstrated that the dog was susceptible to *Rickettsia rickettsii*, the agent of Rocky Mountain spotted fever, and that the organism could be isolated up to the eighth day after inoculation. In two of three dogs challenged, there were no clinical symptoms. In the third, a puppy reacted with fever and respiratory symptoms.

Badger demonstrated in another experiment that dogs may be infected with spotted fever by the bite of infected ticks. Infected *D. andersoni* were allowed to feed on two puppies. Both puppies reacted to the feeding and the virus was recovered. The strains of Rickettsiae isolated from the dogs produced reactions typical of spotted fever in guinea pigs and rabbits, agglutinins for *Bacillus proteus* X_{19} (type O) in rabbits, and complete cross immunity with a known virus of spotted fever. The Weil-Felix reaction with the sera of these dogs was negative, with the exception of one which infected ticks fed on.

In another project, a dog raised in the endemic area was inoculated with infected guinea pig blood. No symptoms were observed, nor were attempts to isolate the Rickettsiae successful. Badger states that apparently this dog was immune to the *R. rickettsii*.

A young sheep was shown to be susceptible. An animal born after the close of the tick season was used so as to rule out any possible acquired immunity. The lamb was inoculated with infected guinea pig blood. After four days, the Rickettsia was recovered and on the following sixth, eighth, and tenth days. The isolated agent produced typical results in guinea pigs. The lamb serum produced a complete agglutination of *B. proteus* X_{19} (type O) in the titer at 1:640.

Topping, in later studies, demonstrated by serological methods that dogs were infected in Long Island, New York, and in Maryland. Dyer has reported that human infections have been acquired from ticks brought into the home by household pets. In other instances, persons have acquired the infection by crushing ticks with their fingers while removing them from dogs.

The disease has also been described by Ortiz in dogs in northern Mexico where large numbers of human cases have occurred. In this case, Ortiz stated that the dogs were febrile with varying symptoms of general malaise. It was reported that a number died.

It is the belief of some public health authorities and veterinarians that the dog, as well as other mammals, is part of the chain of Rocky Mountain spotted fever in nature. There is good evidence that the dog is susceptible, and the virus circulates in its blood. No doubt, infected ticks will produce infection in dogs, and ticks probably can become infected from diseased animals. This also raises a question about the possibility of dogs carrying infection into disease-free areas. It is a problem which will challenge the resourcefulness of public health veterinarians.

Insofar as can be determined, there have been no reports of naturally occurring Rocky Mountain spotted fever in dogs by veterinary medical clinicians or any other investigators. It is strange that no animal cases are reported in the Middle Atlantic and Rocky Mountain States where the infection is endemic. If practitioners desire laboratory assistance in establishing a diagnosis, the Communicable Disease Center Laboratories will be available.

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From the School of Veterinary Medicine of the University of California, Davis, it is indirectly learned that the antibiotic dihydrostreptomycin administered intramuscularly and into quarters infected with *Streptococcus agalactiae* type of mastitis has been giving promising results. The dosage depends on the severity of the symptoms. Dihydrostreptomycin was administered at 12-hour intervals. It is reported that 75% of affected animals in the dry lot responded to this treatment and that they were returned to the milking line as profitable producers. Those that did not respond to this treatment were marketed. The coliform type of infection has not been found to be so responsive to this antibiotic.

PRINCIPLES OF ANIMAL HEALTH

R. R. DYKSTRA, D.V.M.
Manhattan, Kansas

Fringed Tapeworms of Sheep

Up to this time, no success has been attained in attempts to complete the life history of the fringed tapeworm, *Thysanosoma actinoides*. The full life history of the large tapeworm of sheep, *Moniezia expansa*, is well known; it spends a part of its development in the bodies of several species of grass mites. Judging from this, it would naturally be assumed that the fringed tapeworms may have a somewhat comparable life history, but the Zoological Division of the U. S. Bureau of Animal Industry, in its 1951 Report of the Chief, states: "Nearly 84,000 insects, mites, and other forms of small animal life have been collected on pastures grazed by lambs parasitized by the fringed tapeworm", but the most careful "examination and feeding tests yielded consistently negative results." The parasite does not seriously disturb the health of the lambs; the loss results mainly from liver condemnations.

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Ring Test — Valuable Diagnostic Aid

As most of the readers of this column probably recall, the ring-test, also known as the milk test or the ABR test, was officially recognized and made a part of the brucellosis eradication campaign by the United States Livestock Sanitary Association during its sessions in Kansas City in November 1951. The latest figures indicate that during the last four months of 1951, it was used in 11 states to test 49,027 herds representing 810,777 cattle; and that in 1952, it has already been used in 20 states with 454,732 herds and 8,585,476 cattle. If, as a result of the ring test, the presence of brucellosis in the herd is indicated, then the individual blood agglutination test is applied for the detection of the infected members of the herd.

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A news release indicates that three researchers at the South Dakota Agricultural Experiment Station in cooperation with Federal Bureau of Investigation fingerprint experts, have developed a method of classifying bovine noseprints. Up to the present time, it is claimed that they have been able to classify the noseprints of any breed into any one of 5,832 different groups.

Avianized Rabies Vaccine

A recent discovery is that the chick embryo passage of certain strains of rabies virus alters its pathogenicity for mammals. The so-called Flury strain of rabies virus seemingly has lost its pathogenicity for dogs and rabbits when inoculated extraneurally, or it is claimed to be entirely safe, with no neuroparalytic sequelae, when administered intramuscularly into dogs. A longer immunity is said to result because of a high antigenicity. However, injected intracerebrally it will result in rabies. In man, the Pasteur treatment has been followed by some postvaccination paralysis. Though data are incomplete, the hope has been expressed that in man the Flury strain may be as safe and effective as it appears to be in dogs. Thus the avianized vaccine holds hope of freedom from postvaccinal neuroparalytic sequelae in man.

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Terramycin for Avian Salmonellosis

One of the conclusions of Dr. Zaki Morcos of the Department of Bacteriology of the Cairo Veterinary College (*Brit. Vet. Jour.*, Oct. 1952) in regard to the treatment of salmonellosis in fowls is that "Treatment with three successive doses, 250 mg., of terramycin is satisfactory, economic, and scientific." The terramycin is administered in a capsule by way of the mouth, daily for three successive days. Blood clot cultures were negative the day following treatment, and two months later were still negative. Also "Following the seventh day of terramycin application and onwards for two months, cultures from the eggs were negative." The fowl gained in weight and condition during treatment though egg laying was suspended for a few days.

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It is informative to note in a report from the British Royal Veterinary College that sarcoptic mange in a hog was apparently relieved by three hand applications, at intervals of approximately a week or two, of a saturated solution of magnesium sulfate. Condition of the skin was much improved, and no parasites or eggs were demonstrable after treatment. Some of the saturated solution was even poured into the animal's ears with no harmful results.

POULTRY PRACTICE PROBLEMS

W. R. HINSHAW, D.V.M., Ph.D.
Frederick, Maryland

Gizzard Worms

Chickens, turkeys, geese, and ducks are subject to infestation with gizzard nematodes which are widely distributed in North America. The species found in chickens and turkeys is *Cheilospirura hamulosa* (Diesling, 1851) while geese and ducks may become infested with *Amidostonium anseris* (Zeder, 1800). These worms are found beneath the horny lining of the gizzard. In such infestations, the lining of the gizzard of chickens and turkeys may show small local lesions which may extend into the muscular tissue. Nodules enclosing the parasites may be found.

In geese and ducks the lining of the gizzard, if heavily parasitized, appears necrotic, loosened and often sloughed. Adjacent to the site of the worms the tissue is dark brown or black. Grasshoppers, beetles and sandhoppers serve as intermediate hosts for *C. hamulosa,* but *A. anserina* is directly transmissible to its host after a short incubation period outside the body of the host.

Remedies for successful removal of gizzard worms have not been discovered. Prevention consists in rotation of ranges and the usual managerial procedures for handling parasitic diseases.

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Duck Virus Hepatitis

During the spring of 1949, a new and highly fatal disease of young White Pekin ducklings occurred on a number of farms in the duck raising sections of Long Island, New York. Levine and Fabricant (*Cornell Vet.*, 40:70, 1950) made extensive studies of this previously unreported disease and succeeded in determining the cause to be a virus which could be passed through both Seitz and Berkefeld W filters. Levine (Poultry Diseases, by Biester and Schwarte, Ames, Iowa State College Press, ed. 3, 1952, p. 689) describes the disease in detail and reports additional research findings.

The disease has affected ducklings from one to three weeks of age. Onset is rapid with the majority of losses occurring within three to four days. Sick ducklings become listless, fail to keep up with the brood if disturbed and soon become exhausted and sit with their eyes practically closed. After a short time, they fall on their sides, kick spasmodically with both legs and die often within an hour after first symptoms are noted.

The outstanding autopsy finding is the enlarged liver with its punctuate or ecchymotic hemorrhages. Both the liver and spleen may be mottled. Spleen and kidney may also be enlarged. The disease appears to be specific for ducklings since neither turkeys nor chickens exposed have developed it. Diagnosis requires elimination of other infections, isolation of the virus and proof of its nature by serum neutralization tests.

Unsuccessful attempts to produce a satisfactory vaccine have been made by Levine and his coworkers. Serum therapy applied both under laboratory and field conditions, has, however, proved highly successful. This consists of injecting 0.5 ml. of duck virus-hepatitis antiserum (preserved with 0.5% phenol) intramuscularly into all ducklings of a brood when the first few deaths occur. This serum has proved successful in preventing losses in the Long Island duck-rearing area. An antiserum bank, sufficient to treat 3,000,000 ducklings, is maintained at the Duck Disease Laboratory, Long Island, New York. The antiserum for this bank has been obtained by mass collection of blood from exposed and recovered ducks at the time of killing for market at processing plants. This is probably the only antiserum bank maintained for use in a control program for animal diseases.

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Ethylene oxide gas used as a fumigant was found by Lorenz, *et al.*, (*Poultry Sci.*, 29:545, 1950) to be an efficient means of disinfecting shells of eggs which had previously been infected with Pseudomonas sp.

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Tests with lindane vaporizers in a room (11,000 cu. ft.) revealed that even with the doors and windows open, as little as 0.4 gm. of vaporized lindane (less than seven parts in 10,000,000 in the room's atmosphere) killed between 60 and 70% of flies within three hours. With doors and windows closed, practically 100% fly-kill was obtained in the same period. Operation of the air conditioning unit in the otherwise closed room did not affect efficiency of the vaporizers.

Case Report of Unverified Lymphocytic Choriomeningitis in a Chinchilla

WILLIAM T. SHALKOP,* D.V.M., Washington, D.C.

Lymphocytic choriomeningitis occurs in mouse and man as naturally infected hosts; the guinea pig, rat, monkey, and hamster can be infected experimentally but to the writer's knowledge it has not been reported in the chinchilla. This is a case report suggested by histologic findings. Unfortunately material was not retained for animal inoculation and so verification of the condition by demonstration of the virus in laboratory animals could not be made. The purpose of this report is to call attention to the possibility of lymphocytic choriomeningitis occurring as a natural infection in chinchillas. If the symptoms are suggestive of a meningeal involvement, laboratory animals should be inoculated so that if the characteristic pathologic changes[1,2] are found on histologic examination, and the laboratory animals show characteristic histologic lesions there will be verification by animal inoculation.

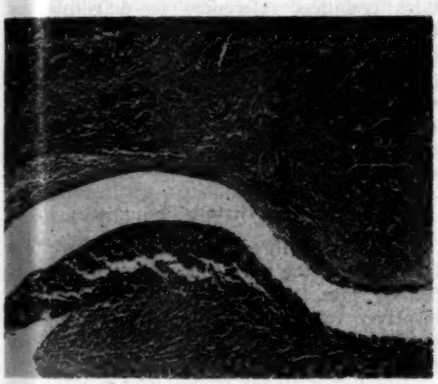

Fig. 1. Choroid plexus of lateral ventricle heavily infiltrated by lymphocytes. Several vessels show perivascular cuffing. X55. Armed Forces Institute of Pathology negative, 524638.

Five animals had been lost over a period of one month in a herd of about 50 chinchillas. The animal made available for this study had been sick about one week and exhibited listlessness, anorexia, and drowsiness. A few days before death it had difficulty in maintaining its balance, falling on its side when walking.

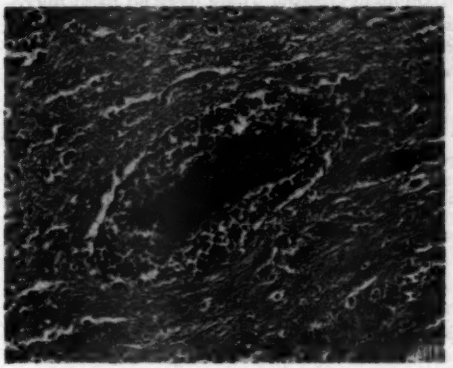

Fig. 2. Lymphocytic perivascular cuffing in cerebrum. X275. Armed Forces Institute of Pathology negative, 524637.

The postmortem examination revealed numerous small necrotic foci in the liver, and the lungs were dark red and edematous with a small amount of clear fluid in the pleural cavity. The spleen was enlarged about three times and showed what was apparently a large hemorrhagic infarct. The other organs were normal in appearance.

Bacteriological cultures of the brain, pleural fluid, liver, and heart blood were negative for aerobes.

The histologic examination showed a severe choriomeningitis and marked perivascular cuffing of the vessels of the brain stem and cerebrum (fig. 1 and 2). The cell infiltration was

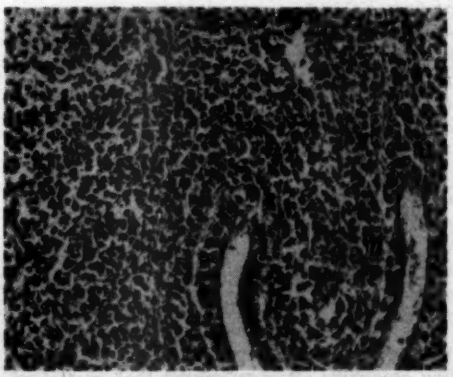

Fig. 3. High magnification of focus in liver to show lymphocytic type of infiltration. X275. Armed Forces Institute of Pathology negative, 524636.

*Pathological Division, Bureau of Animal Industry, U. S. Department of Agriculture, Washington, D. C.

predominantly lymphocytic, although in some areas of the meninges there were a number of macrophages with a large amount of pink-staining cytoplasm. There were no focal microabscesses usually associated with listeriosis. There were also numerous focal accumulations of lymphocytes in the liver where they had replaced the liver cells in these circumscribed areas (fig. 3). The foci showed no predilection for any particular part of the lobule. In the lung there was a heavy peribronchiole lymphocytosis and interstitial infiltration by the same type of cell (fig. 4). In the kidney there was a slight interstitial lymphocytosis, this organ apparently not being severely affected. The infarct in the spleen had involved about one-quarter of this organ, producing coagulation necrosis of all the cells in the affected area. The remaining three-quarters of the spleen showed no remarkable changes.

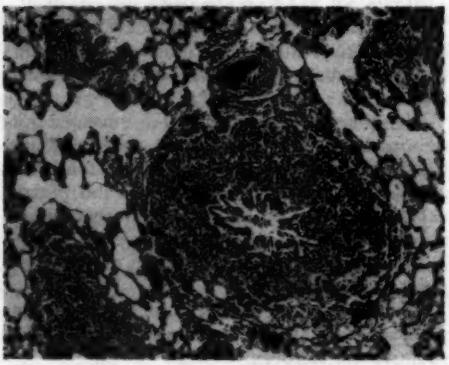

Fig. 4. Peribronchiole and interstitial infiltration in lung by lymphocytes. X75. Armed Forces Institute of Pathology negative, 524631.

After Gram and Giemsa stains of the sections, bacterial forms could not be demonstrated in any of the lesions of the tissue sections.

Considering the inability to recover bacteria from the liver, pleural fluid, heart blood, and brain and considering that the pathological changes are characteristic of a virus invasion, the evidence is strongly in favor of a virus being the cause of the changes; moreover, they are suggestive of the changes observed in lymphocytic choriomeningitis as it occurs in the mouse and experimental animals[1,2,3].

As previously stated, the purpose of this report is to offer histologic evidence that lymphocytic choriomeningitis may naturally affect the chinchilla. It is suggested that animal inoculations be included along with the other laboratory procedures in suspicious cases so that verification may be obtained in case the lesions are similar to those occurring in lymphocytic choriomeningitis in other animals.

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Coliform Mastitis in Swine — A Case Report

C. F. HELMBOLDT,* D.V.M., Ph.D.,
H. H. HALE,* B.S., and J. D. WINN,* B.S., V.S.,
Storrs, Connecticut

Although modern veterinary literature contains countless references to diagnosis and treatment of bovine mastitis, little mention is made of porcine mastitis[a]. The following report indicates that this disease can reach serious proportions even in a well managed swine herd; it further indicates that prompt and intensive treatment can result in a favorable outcome.

History

A 550 lb., three-year-old Yorkshire sow farrowed for the third time with a litter of 12 healthy pigs. Eighteen hours later the sow displayed reluctance to stand, with the result that the herdsman summoned veterinary assistance.

Surroundings and conditions were ideal. The building was of recent construction and clean, and the diet was considered the best available.

Symptoms

The sow presented the picture of an acutely ill animal. Respiration was rapid and the temperature was 106.8 F. Large, irregular, purplish areas up to four inches across covered the ventral surfaces of the body; they blanched under pressure. The three left posterior mammae were markedly indurated and palpation elicited protesting grunts. At this time the sow refused to rise. The herdsman volunteered the information that in the past 45 days two other sows had presented identical objective symptoms and had died in spite of treatment with massive doses of penicillin.

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Treatment

The nipples, areola, and surrounding areas of the three involved glands were scrubbed with 70% alcohol and a few streams of milk were discarded. Milk was then collected in test tubes containing bromocresol purple. The solution turned a greenish hue indicating an alkaline reaction.

Pigs were removed and given reconstituted milk. The sow was given 3,000,000 units of penicillin in oil and 2 gm. of dihydrostreptomycin by the intramuscular route.

Unincubated films of the collected milk were stained by the Prescott and Breed technic[2] and examined. The leukocyte counts were from 2,000,000 to 50,000,000 leukocytes per ml. Blood agar plates were streaked and incubated over night at 37 C. under 10% CO_2 tension. The next day the colonies on each plate resembled those of coliform organisms. Subsequent bacteriologic studies, which included the IMVIC reaction,[1] revealed that the organism was an encapsulated form of *Aerobacter aerogenes*. On this, the second day of illness, no improvement was noted in the sow. Temperature was 105.2 F. Since the mastitis appeared to be due to coliform organisms, the penicillin was discontinued and dihydrostreptomycin was administered intramuscularly at the rate of 2 gm. every six hours. On the third day of illness, the temperature dropped to 103.2 F. and the sow was able but reluctant to rise. The involved mammae were still markedly indurated but the localized congested areas were fading. On the fourth day, the temperature was 103.2 F., the cutaneous congestion had entirely disappeared, and the involved mammae were definitely softer while the normal glands were involuting. Furthermore the sow showed a definite interest in food. The fifth day was the last day of treatment as the temperature remained normal and the sow resented the intramuscular injections. All mammae, involved and normal, were definitely involuting.

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Used crank-case oil drained from farm machine motors is used commonly as an insecticide for swine and cattle or as a vehicle for concentrated insecticidal preparation.

Recent experimental work indicates that such usage of these dirty petroleum products may cause hyperkeratosis (X-disease) in cattle.

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Siamese Twins in a Buffalo Cow (An Abstract)

G. S. CHINNIAH, Colombo, Ceylon

On December 24, 1951, the writer was called to examine a valuable Buffalo cow in a local dairy. The owner represented the animal as restless and in great pain. The following day delivery appeared imminent and on manual examination a transverse position of the fetus was noted with the leg-over-neck attitude.

After considerable difficult manipulation, embryotomy was decided upon and performed, during the course of which evidence of the peculiar twin development was noted. The first calf died during the surgery and, though effort was made to deliver the calves individually, the writer was unable to do so. The twins were inseparable, as may be noted by the accompanying illustration, joined together from flank to flank in a position of trunk presentation. The second calf died a few minutes after delivery.

Repeated injections of penicillin controlled uterine infection. The cow recovered uneventfully in approximately two weeks.

Unusual Case of Rabies in a Holstein Cow

SIDNEY R. NUSBAUM, D.V.M.,
Herkimer, New York

A six-year-old, grade Holstein cow was examined when the owner complained of lessened milk production.

The cow had freshened ten weeks previously. She had cleaned normally but had not exhibited estral signs since calving. Milk production previously had been 60 lb. per day, with the last two milkings just half that amount.

The animal appeared bright, in good condition, and was chewing her cud. Temperature, pulse, respiration, and rumen activity were normal. An area about 1 cm. in diameter appeared to have been scratched or cut in the submaxillary region; the deeper tissues did not seem to be involved. Urine acetone test was negative. Rectal examination disclosed one uterine horn flaccid in tone and slightly distended.

Diagnosis was withheld. It was concluded that pathological changes in the uterus were insufficient cause for such an abrupt drop in milk production; however, 25 mg. of stilbestrol were administered.

The next day the cow still was eating well and appeared bright. Constant tenesmus, with partial eversion of the rectum and vagina, was observed. The owner stated that this condition had started within eight hours of the rectal examination the previous day. A cautious re-examination revealed no obvious damage having been done as a result of the rectal examination. The writer, being careful to wear rubber gloves, administered 1.5 oz. of chloral hydrate by mouth somewhat as an aid in diagnosis. That evening it was reported that this dosage had eased the extreme tenesmus for a few hours. The chloral was repeated.

On the third morning, tenesmus persisted; appetite and general appearance remained normal. There was no motor excitement nor signs of incoordination. The owner reported that the second dose of chloral hydrate did not appear to have had as much effect as the first though the patient had rested and seemed somewhat narcotized. A long lasting local anesthetic was administered epidurally. The cow could not use her rear legs. Tenesmus persisted.

No change was observed on the fourth, fifth, or sixth days, except that milk secretion lessened and the cow began to lose weight. Appetite was noted to be good and general appearance normal.

On the seventh day, periods of hoarse bellowing began. Increased bellowing and decrease of milk production continued until the 12th day. For the next two days, the animal bellowed almost constantly. By this time the cow was practically dry though she ate well.

On the 14th day, paralytic symptoms were noted for the first time. Strangely enough, this animal still was able to chew and to swallow. She went down and died the night of the 14th day of observation, the 15th day after a drop in production had been noticed.

Laboratory examination of the brain revealed the presence of typical Negri bodies.

This case was interesting because of the prolonged course of the disease, the absence of many symptoms considered typical, and the possibility that the examination by the writer was the cause of the tenesmus and thus confused diagnosis until other symptoms (bellowing) developed.

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Listeriosis of Swine

D. F. EVELETH,* Ph.D., D.V.M.,
ALICE I. GOLDSBY,* A.B., and JENNY TURN,*
Fargo, North Dakota

Listeria monocytogenes has been isolated from swine by Slabosptis 'Kii,[1] Biester and Schwarte,[2] de Blieck and Jensen,[3] Kerlin and Graham,[4] Rhoades and Sutherland,[5] and Bolin and Eveleth[6].

Listeriosis is widespread and appears to be of more importance as a cause of animal losses each year. Biester and Schwarte[2] described symptoms and lesions of impairment of the central nervous system in the cases studied by them. Most other investigators have been more inclined to consider the Listeria organism as of secondary importance in swine.

Case 1. The veterinarian and client presented four pigs to the laboratory on May 18, 1949. There were 126 weanling pigs in the drove. The male pigs had been castrated ten days previously. On May 16, several of the pigs had been observed going around in circles. Two died on May 17 and four showing typical symptoms were brought to the laboratory. They had temperatures of 102.4, 100.5, 102.8 and 102.5 F. One pig was

*Department of Veterinary Science, North Dakota Agricultural Experiment Station, Fargo. Published with the permission of the director, North Dakota Agricultural Experiment Station, Fargo.

killed and examined. Gross tissue changes were minor. A few petechial hemorrhages on the heart and some congestion of the mesenteric lymph nodes were observed. Two of the remaining three pigs died and one recovered. *L. monocytogenes* was isolated from the brains of two of the three pigs examined.

Case 2. Eight pigs of 150 died a week following castration. These pigs were observed circling and staggering. When they could no longer stand they lay on their sides and continued to move their legs in running motion. No more cases developed in this drove. *L. monocytogenes* was obtained in culture.

These two cases suggest that either the involved pigs were carriers of the Listeria organism and because of the debilitating effect of castration the organisms became pathogenic or that organisms gained entrance through the castration wounds.

The observations made by Graham, Levine and Morrill[7] indicate that swine can be infected with Listeria by conjunctival exposure.

Several droves of swine have been observed in which symptoms of paralysis were apparent. In all but these two instances the writers have failed to obtain Listeria in culture. Teschen disease has been suspected but attempts at transmission have been unsuccessful.

Information available at this time would indicate that listeriosis is not a prevalent disease of swine but must be considered in the diagnostic procedure in cases in which swine show symptoms of central nervous system involvement.

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Leather can be made from the skins of fish, and from every type of animal from goats to frogs.—*S. N. L.*

Neomycin in the Treatment of Bovine Mastitis

H. M. BRAEUTIGAM, D.V.M.,
Frankenmuth, Michigan

The following article is based upon the value of neomycin in treating clinical cases of mastitis. Diagnosis of mastitis was in all instances based upon microscopic examination of aseptically collected milk samples.

Acute cases of mastitis were treated when first seen. Treatment consisted of infusion of teatube neomycin® into the udder. Three to four tubes per quarter at 12-hour intervals were usually necessary in these hot, swollen quarters. Supportive treatment, using sulfonamides orally or intravenously and penicillin intramuscularly, was employed as the needs of the patient indicated.

Chronic cases of mastitis and noninfectious cases were given local treatment only and seemed to respond favorably with two infusions per quarter at 24-hour interval.

Milk from treated quarters in all cases was discarded for 72 hours.

Summary of Quarters Treated

	Number Treated	Number Infections Overcome	Remarks
Streptococci	46	40 (87 %)	3 quarters dried up
Coliform	34	31 (91.2%)	1 lost function of quarter
Noninfectious	21	19 (90.5%)	2 sold for beef
TOTAL	101	90 (89.1%)	

Of the six streptococcic quarters that did not respond, three responded to penicillin therapy. Various antibiotics were used on the other three without success. Of the three coliform quarters not responding, one was beyond repair, one responded to streptomycin therapy, and the other did not yield to treatment.

The results in treating cases of mastitis indicate this new antibiotic to be an effective and useful treatment aid for this common malady.

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The farm press generally has expressed confidence in the selection of Ezra Taft Benson as the next Secretary of Agriculture. They share the view that he will bring to the job understanding leadership because of his qualifications, by training and experience, and attack the problems of farmer-government relations realistically.

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BOOK REVIEWS

Books reviewed in this department and any other books, if available, may be obtained by remitting the published price to the Book Department, Veterinary Medicine, Suite 803 Livestock Exchange Building, Kansas City 2, Missouri.

DISEASES OF POULTRY, edited by *H. E. Biester*, Professor of Veterinary Research and Associate Director, and *L. H. Schwarte*, Professor of Veterinary Research, both associated at the Veterinary Research Institute, Iowa State College, Ames, with 35 collaborators. Third edition, revised and enlarged. 1,245 pages, 377 illustrations, buckram binding. Iowa State College Press, Ames, Iowa, publishers, 1952.—Price $11.50.

The editors of the first edition of this comprehensive treatise on poultry diseases indicated in their preface that the decision to prepare a book of this character was reached after translating van Heelsbergen's textbook on poultry diseases. Originally they planned to revise van Heelsbergen's book, but decided that the rapid progress made in poultry diseases in the United States merited the publication of an original text by American experts in this field. Success of the two previous editions is evidence that this decision was a wise one. The first edition, edited by Biester and DeVries, and the second by Biester and Schwarte, the present editors, were reviewed in previous issues of VETERINARY MEDICINE[1,2].

Diseases of Poultry is a complete reference work on the subject. Its 41 chapters, written by experts in their respective fields, contain information on all the known diseases. Special chapters are devoted to anatomy, digestion, poultry genetics as related to pathology, avian hematology, principles of disease prevention, nutritive requirements, vitamins, and diseases of the turkey.

The third edition is not merely a rewriting of the former editions but a complete revision. The size, 1,245 pages with 377 illustrations, represents 91 additional pages and 19 additional illustrations over the second edition; 240 additional pages and 53 additional illustrations over the original published in 1943. These additions represent the studies made in the field of avian pathology during the elapsed time between revisions. As one of the collaborators, the reviewer realizes the attempts made by the editors to condense the size of the revised third edition without detraction from its value. No doubt, deletions could have been made, but in most instances it would have been by removing essential information.

The death of two noted poultry disease research workers, Dr. J. R. Beach and Dr. L. D. Bushnell necessitated the allotment of chapters previously written by them to other equally qualified investigators. One new chapter, Duck Virus Hepatitis, illustrates the reporting of recent research work included in this revision.

The book is written by research workers and naturally tends to fundamental discussions of the subjects, but practical information on methods of diagnosis, prevention, and control are by no means neglected. Each of the authors has been especially conscious of the recent developments in the use of antibiotics, sulfonamides and other remedies when bringing his section up to date. In the newly written chapter on Newcastle disease, Dr. C. A. Brandly has made an outstanding contribution in his up-to-the-minute review of this important disease. A five-page discussion on the subject of prevention and control of the disease is included for the practitioner. Dr. K. F. Meyer's revised chapter on ornithosis and psittacosis is another example of a complete fundamental and yet practical resume of a subject increasingly important to the veterinarian.

This book is one of the classics in veterinary literature. Its composition, printing and binding are excellent. The contents, with the extensive bibliographies and exhaustive index, make it an encyclopedia of poultry disease information. It is scientifically written for the reference shelf of students, teachers, research workers, practitioners and in fact for everyone concerned with poultry disease investigation, prevention, and control.—*W.R.H.*

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Annual milk production in the United States is now at a record low in relation to the needs of the rapidly increasing population.—*Annual Rept., Bureau of Dairy Industry, USDA.*

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Antibiotic mist produced by nebulizing penicillin and streptomycin in water solution with oxygen is finding some favor for the treatment of cough in dogs when etiology is obscure.

[1] *Vet. Med.*, 38:319 (Aug.), 1943.
[2] *Vet. Med.*, 43:491 (Nov.), 1948.

P.O.P.

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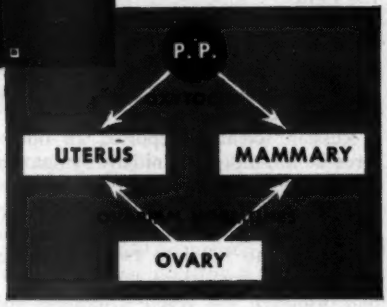

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ABSTRACTS OF LITERATURE

Aoeski Disease[1]

Sporadic appearances of a peculiar central nervous disturbance claimed to be a separate entity have been reported in Russian veterinary literature. The disorder is frequently confused with rabies, epilepsy, poisoning, and nutritional deficiencies. It was named Aoeski disease in 1938 by Sherstaboev and Gaklin (*Veterinaria*, 12, 1948).

Reports have been concerned largely with incidence of the disease in swine, although it has been observed in cattle, sheep, and cats. The infective agent (not indicated) is transferable to laboratory animals.

Russian scientists reported an outbreak in three-month-old pigs. Initially symptoms noted were inappetence, slight rise in temperature, profuse salivation, and an apparent desire to vomit. As the disorder progressed, animals were noted to fall into two categories; those manifesting convulsions and a smaller group that displayed less severe symptoms and were classed as dull. Death followed in either case.

Later, these same workers reported fatal disease in cats and infection in pigs on the same premises. Symptoms described included refusal to eat, involuntary movements, copious salivation, exophthalmia, and hyperemia. Autopsy lesions revealed cyanosis of mucous membranes of eyes, mouth, and nose, hyperemia of lung tissue with accompanying changes in the bronchi, mucosal hemorrhages, catarrhal enteritis, and injected lymphatics.

Other reporters (Lukashov and Nikitin) observed the malady among sheep, pigs, and a heifer. In these, intense itching about the head, mouth, and lips was a prominent symptom, as was a nasal discharge. Excitement followed by exhaustion is stated to be responsible for deaths.

No mention is made of the character of the etiological agent except that bacterial cultures proved negative. Suspension of tissue from diseased animals when injected into rabbits resulted in rapid death. Bacterial cultures propagated from these laboratory animals also gave negative results.

Symptoms of this disease parallel those observed in a few swine droves in this country, though this is not to say the disease exists here. Complicated nervous disorders need further study to differentiate and classify those fatal diseases in which nervous symptoms predominate.

[1]Merlen, R. H. A., M.R.C.V.S., Aoeski Disease. *Brit. Vet. Jour.*, 108:441 (Nov.), 1952.

Benzethacil — A New Penicillin

The necessity for frequent administration of penicillin to maintain effective blood levels of the antibiotic in the early use of this therapeutic agent was the most undesirable feature. Demand for a vehicle or product that would maintain sufficiently high blood levels for longer than three or four hours stimulated research beyond the hopes of clinicians who employed penicillin shortly after the close of World War II.

Water soluble penicillin, first made available, was rapidly eliminated by the kidney; later, oils and aluminum monostearate were employed as vehicles to slow absorption. Other effort was made to prevent elimination by structural changes of the molecule containing the antibiotic.

Most recent advance in the prolongation of blood levels known to be effective is the development by the Wyeth Institute of Applied Biochemistry of benzethacil, chemically N, N¹-dibenzylethylenediamine dipenicillin G. Administration of a single intramuscular injection of 1,500,000 units of this compound to cattle of various ages resulted in effective blood serum levels of six to nine days' duration[2].

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Vesicular Exanthema Quarantine Lifted in Three States

Federal quarantine restrictions imposed because of vesicular exanthema have been removed in Illinois, Ohio, and Connecticut. However, the shifting of infection to new areas is noted by the report that part of Tennessee has been added for the first time as a quarantine area because of this disease in swine.

Nine counties in New Jersey, all in Rhode Island, and all except two in California remain under restriction. In these, no action is reported of generally applied control measures though much material has been submitted for legislative consideration in a number of states.

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Accurate field diagnosis of respiratory diseases in poultry without resort to laboratory tests is not now possible.

[2]Huebner, R. A., Bovine Penicillin Blood Levels Obtained with Parenteral Benzethacil. *Cornell Vet.*, 42:457 (Oct.), 1952.